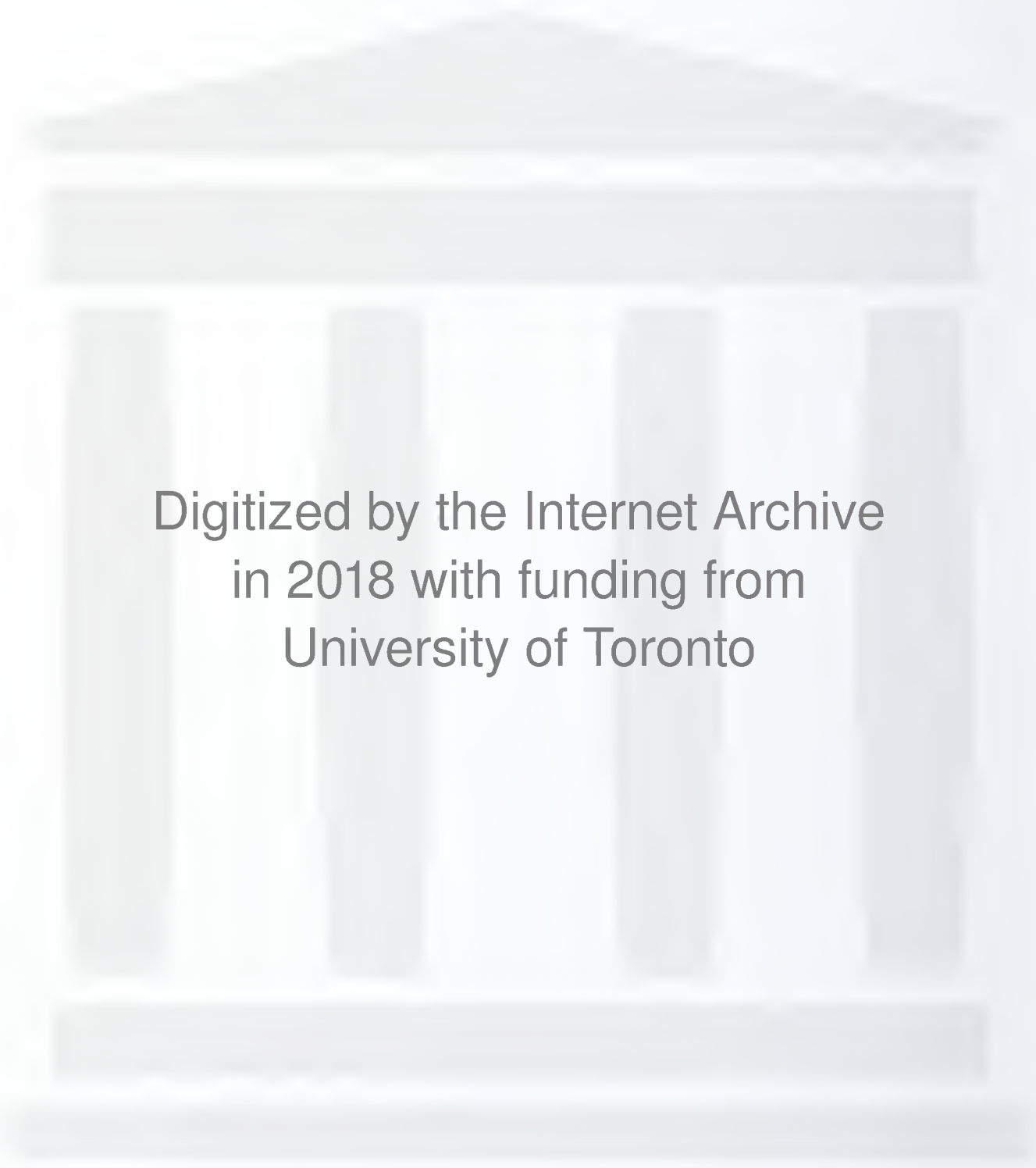




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Editorial

On Clinics

It is hoped that the remarks which are to be made on the subject of clinics will not be construed as carping criticism. That the University of Toronto has one of the finest medical schools on the continent, if not in the world, there is no doubt. However, nothing is so perfect that it cannot be improved, and in our clinical years, we have observed one or two things which we think could be changed with advantage to all.

The clinic to which we refer is the one in which a group of students is conducted by a clinician to the bedside of a patient. There, an examination of the patient is followed by a discussion of his disease in most of its aspects. There are two criticisms, first, the discussion of the disease in the patient's hearing and second, the long periods of standing for the students.

As to the first, one of the axioms which was impressed on us early in our study of medicine is "The patient is a person." It seems that this is sometimes forgotten in the clinics where the patient's symptoms, all his possible diagnoses

and his prognosis are discussed around him. Often discussion shifts to more serious conditions, and a chance remark on these or his own disease may be seized upon and have a profoundly detrimental effect on him.

The second criticism may seem sheer laziness. Nevertheless, it is a physical strain to stand for periods of from half an hour to one and a half hours. When one considers that it would be quite possible for the examination to be completed in a few minutes, and for the students and clinician then to retire more comfortable regions to have the discussion, the strain is seen to be wasted and useless effort. There is also a considerably different point of view. When one's feet are aching, and one is shifting from one to the other, and trying to balance in an upright position, concentration on what the clinician has to say is impaired, often to the degree of complete inattention.

So much for the criticism. Our suggestion is that students and clinician examine the patient, learn all that is to be learned from him, then retire to some place outside his hearing where all can enjoy the comfort of a seat.



Old Way . . .

CURING RICKETS in the CLEFT of an ASH TREE

FOR many centuries,—and apparently down to the present time, even in this country—ricketic children have been passed through a cleft ash tree to cure them of their rickets, and thenceforth a sympathetic relationship was supposed to exist between them and the tree.

Frazer* states that the ordinary mode of effecting the cure is to split a young ash sapling longitudinally for a few feet and pass the child, naked, either three times or three times three through the fissure at sunrise. In the West of England, it is said the passage must be “against the sun.” As soon as the ceremony is performed, the tree is bound tightly up and the fissure plastered over with mud or clay. The belief is that just as the cleft in the tree will be healed, so the child’s body will be healed, but that if the rift in the tree remains open, the deformity in the child will remain, too, and if the tree were to die, the death of the child would surely follow.



It is ironical that the practice of attempting to cure rickets by holding the child in the cleft of an ash tree was associated with the rising of the sun, the light of which we now know is in itself one of Nature’s specifics.

*Frazer, J. G.: The Golden Bough, vol. 1, New York, Macmillan & Co., 1923

New Way . . .

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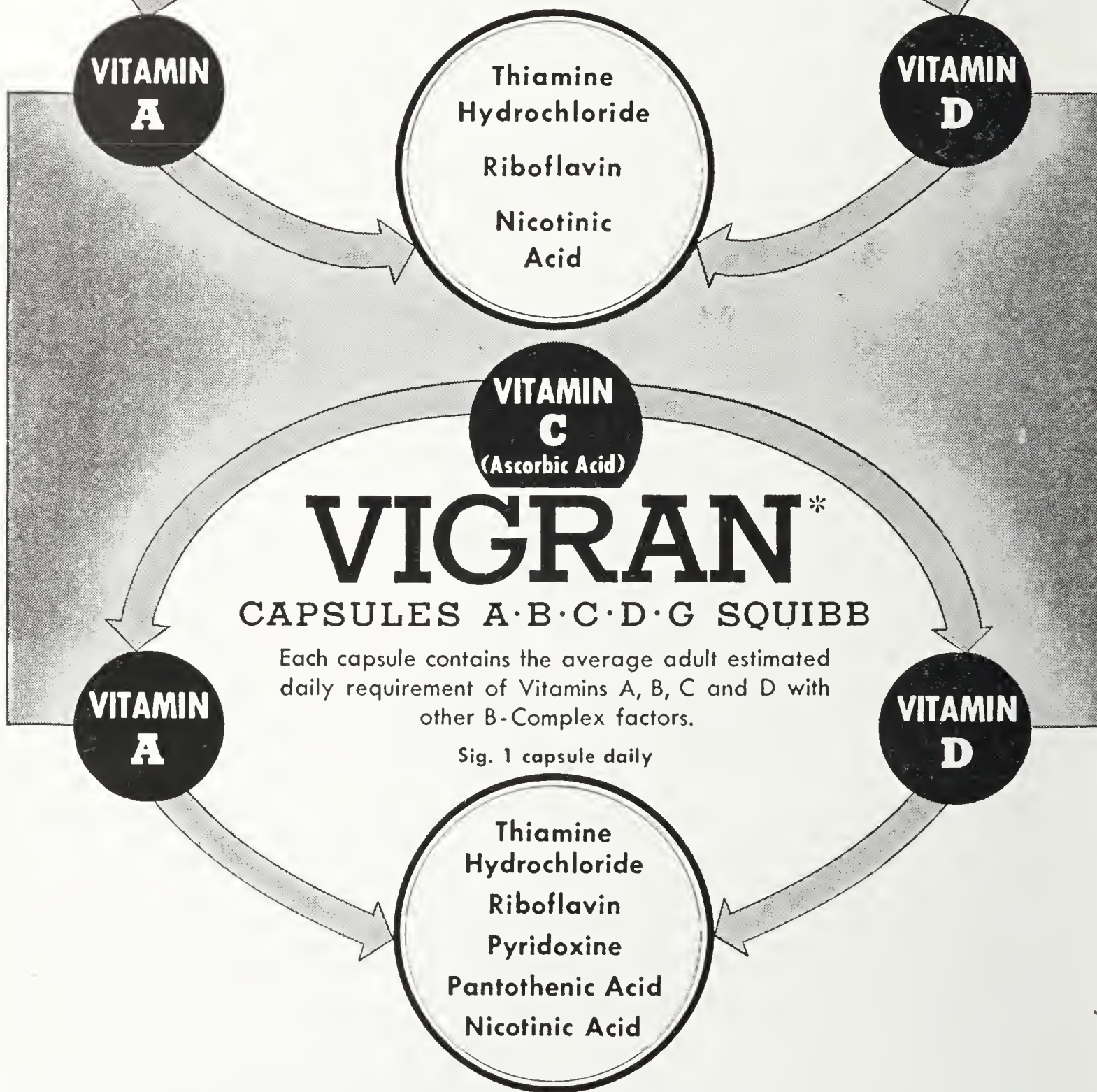
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COMPOUND FRACTURES

Part II: A Review and Criticism

By W. E. GALLIE, M.D., F.R.C.S.(Eng.), F.R.C.S.(C.)
*Professor of Surgery and Dean of the Faculty of Medicine,
University of Toronto*

So much has been said and written in the past few months on the treatment of wounds resulting from enemy action that a discussion of that subject would be a mere repetition. It is worthy of note, however, that progress in the treatment of wounds in civilian practice always comes from experience in war, so that it is to be expected that the new suggestions that are being tried out now on the battlefields of Europe will have an important influence on our methods of treatment in the future.

My attention to the importance of keeping one's feet on the ground at times when all sorts of new ideas are appearing in the Medical journals was sharply attracted by the answers given by the candidates at the recent final examinations of the Medical Council of Canada. The question I have in mind was, "In a road accident a man sustains a compound fracture of the tibia and fibula midway between ankle and knee. Detail your treatment from first aid to the conclusion of the case". Now I do not know whether the gentlemen who set the paper, Drs. Barlow and Malcolm Cameron, were thinking of the wounds sustained in the roads of Coventry during an air raid or of the ordinary compound fractures of the leg that are brought in to our emergency departments following motor car accidents. If it was the latter, I venture to surmise that they will be astonished with the answers.

Regarding the "first aid" the answers were admirable and reflected the influence of the instruction that has been general since the outbreak of war. Emphasis was placed on the importance of covering the wound with a clean dressing, of preventing the protruding contaminated bone fragments from being retracted into the wound, of immobilizing the fracture

with some improvised splint and so on. One candidate quoted an epigram of his teacher to the effect that it was better to fit a splint to the limb as it lay than to have to fit a coffin to him later on.

It was with the treatment of the wound and the fracture that I found fault. Fully 50% of the candidates stated that following the adequate sterilization of the skin and an exhaustive débridement, the wound should be packed with vaselined gauze and a skin fitting plaster cast applied directly to the skin and the wound. This cast was not split or fenestrated and was left untouched for several weeks, or until the stench became intolerable. When it was removed one could expect union of the fracture and healing of the wound. No mention was made of the familiar complications of osteomyelitis and non-union.

In this answer one recognizes at once the influence of the newspapers and such journals as "Life" and "Time". It is based on the writings of Orr and Trueta and, being new and interesting, it is seized upon by the novice and accepted without question or discrimination. And yet, how many of us use the method? My enquiries have shown that there has never been a case so treated in the Toronto General Hospital and that no one of the teachers has even suggested it.

It has occurred to me, therefore, that it might be profitable for us to review our methods of dealing with wounds and compound fractures in civilian life and to consider wherein the retention of that which is old and the adoption of that which is new may be helpful in the treatment of our patients.

Today as always, the most important part in the treatment of wounds is the prevention of infection. If infection develops, be it streptococcus, staphylo-

coccus, gas bacillus or tetanus bacillus, the course of the disease and the prognosis are so profoundly changed that we are justified in going to all possible extremes to prevent it.

Knowing as we do that the element of time is of such paramount importance in the prevention of infection, are we doing all we can to see that these accident cases come under treatment at the earliest possible moment? I suggest to you that we are not. Right in our cities and towns it is sometimes hours between the time the accident occurred and the commencement of the operation in hospital. Profound shock may, of course, delay matters but most accident cases are not suffering from severe shock, or at any rate shock that cannot be easily controlled by ordinary measures. It seems to me that it is our duty, whether we are doctors practising in cities, towns, mining villages or in the country, to see that all patients with potentially infected wounds or compound fractures be rushed to hospital at once and that action be taken there immediately. I have no patience whatever with emergency services that allow such patients to lie about for hours while operating rooms are being prepared. It is just that delay that so often makes the difference between the aseptic healing of a wound and a ghastly infection.

One of the most dreaded infections in the war of 1914 was that caused by the streptococcus. From it came spreading lymphangitis, gangrene and septicaemia, with an all too frequent fatal result. Until recently it was supposed that this infection was the result of the contamination of the wound from the skin or clothing of the patient or from the projectile. Extensive studies on streptococci, however, particularly by Colebrook and Hare, have shown that it is rare to find pathogenic streptococci in the wounds soon after the accident or even in the patient's clothes or on the projectile. They are of the opinion that when streptococcal infection supervenes, the organisms have been introduced from the air which has been contaminated

with bacteria from the throats and noses of infected nurses or doctors or from the wounds of infected patients. That such delayed infection of a wound is possible is well demonstrated in the appearance of green pus where frequent dressings have been done for ten or twelve days.

The papers on this subject published by Hare have impressed on me more than anything else the great importance of protecting these patients from needless infection with streptococci. Thus in the "first aid" we should emphasize the wickedness of applying a handkerchief to a wound. Nobody with a cold should be allowed near the patient. Nothing should be done to the wound beyond covering it with a sterile dressing if such is available. When the patient arrives in the operating room the same precautions should be taken there as would be taken in a so-called clean operation on a joint or the brain; the same meticulous care with cap, masks and gloves, the draping and toweling of the parts and the changing of gloves and towels as they become soiled. Whereas in the casualty clearing station or advanced dressing station speed may be the prime requisite to maintaining a low mortality rate there is no excuse for it in civilian practice.

Further, the studies of Hare have indicated the importance of observing similar precautions during the subsequent dressings of the wound. Of what value is it to preserve a compound fracture from infection with streptococci if we are going to abandon all aseptic precautions a few days later when the first dressing is performed? And yet I venture to state that in most of our hospitals these dressings are done without gloves or masks and the soiled packs and pads are replaced from dressing jars and packing tubes which are used for one patient after another and must certainly aid in spreading infection. The remedy for all this of course is simple although somewhat expensive. It consists of making all preparations ahead of time for each dressing and having everything needed in the dressing prepared in

a single container, such as arage tray, and put through the sterilizer and kept sterile till required. When this sterile setup has once been opened it goes back again to the sterilizer.

Looking back over the years to 1914 one would say that the outstanding contribution to the treatment of war wounds has been the débridement. This started first with the simple excision of the edges of the wound and then gradually involved the enlargement of the wound to enable the operator to explore all the damaged area and to excise all tissues that were contaminated or of doubtful viability. This operation has now become one of considerable magnitude and calls for a practical knowledge of anatomy, and a sound surgical training. The operation involves excision of the edges of the wound in the skin, all devitalized muscle, all dirt, particles of clothing, bits of loose bone and the projectile, but preserving the main vessels, the nerves and the skin. I am convinced that if it is well done there will be a great reduction in mortality and in morbidity from tetanus, gas gangrene and pyogenic infection, whereas if it is unskilfully or carelessly performed, it will be quite useless.

While this is the recognized primary treatment for shell wounds and for the wounds occurring in the bombing raids it is by no means the accepted plan for the wounds of civilian practice. It was here that I disagreed with my students in their answer to the question on compound fracture. The great majority of compound fractures of the leg occurring in civilian practice are nothing more than simple fractures in which the end of one of the fragments has been pushed through the skin. Frequently the patient arrives in hospital with the end of the bone still protruding. Obviously for such patients anything akin to a wartime débridement would be unnecessary. All that is needed is the careful aseptic and antiseptic cleaning of the skin, the thorough washing of the protruding bone with soap and water, gasoline and saline solution, the excision of the edges of the

wound, the reduction of the fracture and the application of the splint. Some judgment is required in deciding whether to close the wound or not. If one is confident that all contamination has been eliminated, then he should close the wound. If, on the other hand, there is any doubt about it, the wound should be left open so that all discharges may escape. With this plan of treatment for compound fracture, I understand that the last 30 consecutive cases admitted to the emergency department at the General Hospital have escaped infection.

This question of primary suture is one that cannot be dismissed lightly. Undoubtedly the method is attractive as it gets rid of discharges, odours and the risks of subsequent infection resulting from dressings. On the other hand if infection does occur, it aggravates the whole picture by the retention of the infected exudates. It seems to me that it can be employed only in the minor cases and should never be used in anything resembling a war wound.

The question of the role of antiseptics in the prevention of infection is still controversial. We can all remember the enthusiasm with which we hailed the introduction of iodoform, mercurochrome, dichloramine T, acriflavine and other antiseptics and saw them one by one tossed into the discard. It would appear that antiseptics that are strong enough to prevent infection do serious damage to the surfaces of the wound. The plan that had the greatest vogue for a time during and after the old war was that of Carrel and Dakin, using sodium hypochlorite. Even this, however, has disappeared except in a few isolated clinics, the chief of which is that of William O'Neill Sherman of Pittsburg. And now we are in the midst of another whirl of enthusiasm for the local application of sulfonamide drugs. Frankly, I do not know whether they are any good or not. I hope they are, but the younger generation must excuse us, who have had so many disappointments, if we have some doubts about them. The only way to settle the matter would be to set

up an experimental station in England where treatment with and without these drugs could be observed in equal numbers of patients.

The question also of the value of these drugs when administered orally or intravenously in preventing infection in wounds is still quite unsettled. It is certainly attractive to think that by raising the concentration of such a drug as sulphathiosol in the blood, one may be able to prevent bacteria in a wound from causing either local or general infection. No such result has been demonstrated yet, however, so that judgment must be reserved for some months longer. This also is a matter that could be solved by study in an experimental station.

The most revolutionary suggestion that has been presented in a long time is that coming from Barcelona and which is known as the Orr or Trueta treatment. It consists of:

(a) The thorough débridement and cleansing of the wound after opening it widely.

(b) Packing the wound wide open with dry or vaselined gauze.

(c) Complete immobilization of the parts.

(d) Very infrequent changes of dressings.

These are principles that have been advocated by others and they undoubtedly have their virtues. It is claimed, for instance, that covering the wound with sterile dressing and leaving it untouched for several weeks, in spite of the horrible smell that often develops, prevents the introduction of such organisms as streptococci. Again it is taught that the complete immobilization of the part reduces the risk of infection and promotes the healing. Allowing for the over enthusiasm with which new methods are often received, I am convinced that these suggestions have something in them. Years ago, Orr suggested that in operations for the removal of sequestra and the saucerization of cavities the wound should be loosely packed with vaselined gauze and covered with a

plaster bandage which is left untouched for several weeks or until the odour becomes unbearable. At first this suggestion was received with jeers and many a crack about the smell, but in spite of this the principle of infrequent dressings is thoroughly established and is accepted all over the world. From this method of handling chronic osteomyelitis the principle was applied to operations for septic non-union in fractures. And now it is being advocated as a primary treatment of all wounds and particularly for compound fractures.

The question of whether the incidence of infection will be as great in this war as in the last, is something we cannot predict at the moment. Sherman, who is the great advocate of the Carrel-Dakin method, thinks the Orr Trueta treatment is a form of malpractice and maintains that the salvation of the wounded is to be found in keeping the débrided wound flooded with sodium hypochlorite. He quotes an extraordinarily low incidence of infection at the steel plants at Pittsburgh. On the other hand, Trueta records 1,008 cases of compound fracture of the femur with 6 deaths, a figure that was never approached in the last war. The chances are, therefore, that the controversy will go on for some time longer.

As my chief concern in this paper is not with war injuries but with the ordinary accidents that occur in civilian life, I must point out that it is comparatively rarely that the Trueta plan is indicated. As stated at the beginning, I quarrelled with the answers of the students who said that the Trueta treatment was the only treatment and who overlooked the fact that better and quicker results can be obtained by other methods.

An interesting detail in many of the answers was the recommendation that after the débridement all severed tendons and nerves should be repaired. This, of course, is somewhat controversial and calls for good surgical judgment. A good rule to apply is that the repair of tendons and nerves should be carried

out only if the wound is clean-cut as in an accident with glass, if it is evident that there is a high probability of primary union and if the condition of the patient is such as to justify the necessary prolongation of the operation. If there is the slightest probability of infection, however, no such operation should be undertaken as they are not only futile but injurious. I never do more than pass a fine catgut suture through the sheath of the severed nerve or through the ends of the tendon so as to make sure that the ends will be near one another when the day for the formal operation arrives.

My chief quarrel with students, however, had less to do with treatment of the wound than with the treatment of the fracture. The glib recommendation that after the débridement and packing of the wound a plaster be applied directly to the skin without padding of any kind, was more than I could stand. I have seen trouble enough with the padded plasters without deliberately asking for it by applying a skin tight, unpadded one before the swelling has taken place. My strongest advice to you is, don't do it.

To apply a tight plaster on any limb that we know is going to swell is certain, sooner or later, to carry us to the malpractice court. I can hear the sweet tones of the lawyer for the plaintiff when he says, "But doctor, is it not a well-known fact that it is dangerous to apply any encircling plaster cast on a fracture before the swelling has taken place?" And if the answer is not wholly satisfactory, he proceeds to read something out of one of the best known textbooks and tries to prove gross ignorance as well as negligence. I know, of course, the argument that by applying these plasters on the skin before the swelling occurs, the swelling will be prevented and that owing to the presence of an open wound the tendency to swelling will be less. In spite of this, however, the risk of interfering with the circulation is so great that it is, as far as I am concerned, unjustifiable.

These remarks should not be interpreted as meaning that skin-fitting plasters should never be used under any circumstances. While I do not make much use of them, yet I have seen them used in our fracture clinic most successfully. It is only in cases, however, in which the swelling has already occurred or has actually subsided; never before the swelling has reached its height.

It is curious the way this idea that skin-fitting plasters will hold the fragments in position after reduction, has spread. Those of us who have had the most experience with plaster know that no matter how skilfully a spica is applied it will seldom prevent gross shortening of the femur even when the fracture is squarely transverse and the fragments placed end to end in the reduction. It has been my experience that almost invariably the fragments have slipped apart and overlapped within half an hour of the cessation of the anaesthetic. This is even more true when the fracture is oblique or when it is situated in the lower or the upper third. In the case of the leg, while the tendency to overlap is less and the result, when it occurs, less disastrous, nevertheless the final result is often most disappointing.

I do not propose to discuss today the whole field of fractures but I must remind you that out of the last war came one clear-cut lesson, namely, that to maintain length and alignment you must provide, first, a good splint and second, efficient traction. What is the sense, therefore, of abandoning what we learned at such cost, and adopting so credulously what our own experience has taught us is nonsense.

Returning to the compound fracture of both bones of the leg, therefore, let us consider what may be done with the fracture. Some surgeons do not hesitate, after the complete cleansing of the wound, to apply a steel plate and over all a plaster. Others, chief of whom is Sherman, apply a plate and then with the limb in a modified Thomas Splint, institute the Carrel-Dakin technique. And now I hear that some are combining

the Orr-Trueta plan with a steel plate. None of these plans has appealed to us in Toronto, however, because of our fear of causing osteomyelitis. But I must admit that some surgeons do use the method and if infection does not occur it has obvious advantages.

A method much in vogue in Toronto is to deal with the fracture after dressing the wound, as if it were simple, reducing it by manual traction and then applying a plaster with the knee flexed to a right angle and the foot in equinus, so as to get rid of the effect of muscle contraction. This works very well in fractures where the ends of the fragments can be engaged and where infection of the wound is not serious.

The plan that appeals to me most, however, is one that combines an efficient splint with traction. The former is provided by a padded plaster extending from toes to upper part of thigh, with foot at a right angle and knee at 135° ; the latter by means of a Kirschner wire passed through os calcis or lower end of tibia. With the leg resting on a Braun splint ten or fifteen pounds traction is applied and the possibility of shortening, twisting or angulation pretty well eliminated. I make it an absolute rule to cut a strip out of the whole length of the front of the plaster, about 1" wide, and to cut the flannel bandages with a scissors. This precaution lessens the risks of gangrene or Volkmann's paralysis.

The question of when the wound shall be dressed depends on the patient's condition. If his temperature is normal, by all means leave things alone. If, on the other hand, the dressing of the wound becomes urgent, a window can be cut without destroying the splint or the traction and without an anaesthetic.

A word should be said about the necessary precautions against tetanus and gas bacillus infection. Probably the most powerful preventive of these dreaded infections is the primary cleansing and débridement of the wound in which most of the organisms are removed and the dead tissue on which they thrive, excised.

In all probability, too, the wide open packing of the wound in the serious cases allows the air into all recesses and prevents the conditions necessary to the growth of anaerobic organisms. However, we still have confidence in the administration of the prophylactic dose of tetanus antitoxin for civilian cases and even more in the use of toxoid for the soldiers. As far as gas infection is concerned, we doubt the value of the antitoxin and trust more to the local treatment of the wound and the administration of the sulphonamides.

And now for my final complaint about the answer to the question on the paper. Of the 125 candidates only two mentioned the possibility of osteomyelitis or of non-union. Most of the others seemed to have the utmost confidence that the measures they had taken would lead to union of the fracture and the healing of the wound in three or four months. And of the two who mentioned these complications, one said he would remove the sequestra and insert a bone graft!

Osteomyelitis is, of course, a common complication of compound fracture and one that greatly prolongs the convalescence. Healing cannot occur until the sequestra have separated and have been removed and this requires months. It does not necessarily cause non-union for in the presence of the mild sepsis that persists around the necrotic bone, osteogenesis and callus formation seems to be actually stimulated. If the fragments are separated by necrotic bone, however, non-union is common.

Here is the place, par excellence, for the Orr treatment. When the sequestra ultimately loosen, the wound should be opened up, the sequestra removed, the position of the bones adjusted and the wound packed wide open with gauze. Vaseline or Bipp on the gauze help the drainage. Then with adequate traction, probably by means of a Kirschner wire, and an encasing plaster, the patient is put aside like an old cheese to ripen. Six or eight weeks later, when the cast is removed, the fracture will in many instances be solidly united and the wound on the way to closure.

The treatment of non-union after healing of the wound has occurred does not concern us here. If there never was sepsis in the wound the necessary bonegraft operation can be undertaken within a month or so of the healing of the wound. If sepsis was present, however, no bonegraft operation should be undertaken for at least a year after the disappearance of all sepsis. Even then there are risks but they must be taken.

And now this brings us to the end of our examination for the diploma of the Medical Council of Canada. If any of the candidates had been as long winded as I have been, I would have plucked him at once but fortunately they were not. The question they were asked, however, was a simple one and one that we must all face in every day practice. In brief, the answer is as follows:—

1. First Aid: Cover wound with sterile dressing. Apply splint or pillow without reducing the fracture or letting the protruding fragment slip back under the skin. Apply tourniquet if haemorrhage. Give morphia. Transport to hospital at once.

2. At hospital, do a quick physical examination—Treat shock.

3. Operation: Thorough cleansing of limb, wound and protruding bones. Excise edges of wound and if the laceration is a severe one, do a complete débridement. Close wound if small and likely to heal by primary union. Pack with vaselined gauze and leave wide open if at all suspicious. Sulphanilamide can be dusted in if you think it is useful.

4. Reduce fracture and immobilize: I would use a combination of traction with a Kirschner wire and a plaster extending from toes to upper part of thigh with foot at right angle and knee at 135° . Split the plaster and the flannel and spring open enough to eliminate risk of strangulation.

5. Administer 1,500 units of tetanus antitoxin after testing for sensitivity.

6. Administer sulfonamide by mouth if sepsis feared.

7. If temperature falls to normal in a few days and pain is absent, leave things alone for a month. Then remove plaster, dress wound and apply a plaster that will allow the patient to walk on crutches.

8. If sepsis develops, I would open the plaster and remove the packs and institute Carrel-Dakin technique.

COMPOUND FRACTURES

Part III: Principles of Treatment

By R. I. HARRIS, M.B., F.R.C.S.(C.)

Chief of Orthopaedic and Fracture Service, Toronto General Hospital

The modern treatment of compound fractures dates from the War of 1914-1918. In that struggle we were burdened with the gigantic task of devising efficient means of treatment for wounds such as had never been seen in the world before and in unprecedented numbers. Of all the war wounds, those which were complicated by compound fractures were the worst. The measure of our surgical success in that war was the lowered mortality rate and improved function which we finally achieved by the improved treatment of compound fractures. Out of that surgical problem certain fundamental principles of pathology and of treatment emerged, so important and still so applicable that it is worth our while to review them briefly.

When the great war commenced in 1914, surgery for a generation had been aseptic surgery. The sepsis of pre-Listerian days was but a hazy memory of stories told by our fathers and our preceptors. Sepsis as a major problem hardly existed in surgery immediately prior to the Great War. The Wars which immediately preceded gave no hint of the problem we would have to face. The South African War and the Russo-Japanese War did not present serious problems of infection. The small wounds of rifle bullets, the dry and sunny climate of the veldt made infection of minor importance. When the Great War started, circumstances had changed. For the first time in history artillery was used in overwhelming amount. Shell wounds predominated and the extensive lacerations produced by shell fragments and the massive pulping of the tissues along the track of the wound introduced a new kind of wound problem. Moreover, the battles were fought over ground which had been cultivated for centuries and every grain of it contained countless organisms. The treatment of these wounds

by the then current aseptic methods was followed by massive and fulminating infections. The realization was quickly reached that new and more vigorous and more effective measures must be found. The cry at first was "back to Lister" which meant the treatment of the wound by antiseptics. This was but feebly effective. Slowly there evolved the appreciation that the wounds produced by shell fire were unique in two respects; first, the damage to soft tissues in the track of the missile was so great that much tissue was dead; much so enfeebled in vitality that it could not resist infection; second, all wounds were heavily seeded with infection. As these fundamentals became clear it was not long until rational forms of treatment were devised to meet them. Debridement, Carrel-Dakin irrigation and secondary suture became the standard forms of treatment and mortality dropped to new low levels.

The compound fractures produced by war wounds represent in the most extreme degree the problems of compound fractures in civil and industrial surgery. The difference is of degree and not of principle. What we learned of the treatment of war wounds in the last war is the basis of our present treatment of compound fractures. To-day, though we have new therapeutic agents to help us, the fundamental problems remain the same and the principles of treatment also remain unchanged.

Fundamental Problems

The basic difference between simple and compound fractures is the problem of infection. Infection is a major problem in compound fractures for four reasons. First, of course, because the wound is open to the exterior and lacks the protection of an intact skin; second, the trauma of the accident has caused more or less damage to soft tissues so

that some are dead and some devitalized. Dead and devitalized tissues cannot resist bacteria by the natural mechanisms of defense. On the contrary, dead tissue provides an ideal medium for bacterial growth. Third, the circumstances of the accident drive bacteria into the wound. Fourth, the daily exposure of the wound for dressing provides an opportunity for secondary implantation of haemolytic streptococci from the nasopharynx of attendants. In compound fractures, we are faced with the problem of the fracture plus the very serious problem of infection implanted under very special circumstances.

When we operate upon a bone as, for instance, in performing a bone-graft, or an osteotomy, we produce, in a sense, a compound fracture. Yet we expect the wound to heal by first intention and almost invariably it does. What are the reasons for this great difference between operative wounds on bone and compound fractures? There are two. First, by aseptic technique we take pains to minimize the number of bacteria which are introduced into the wound. We may believe that our aseptic precautions completely prevent the entry of organisms into the wound. This, however, is too optimistic. One has only to expose a Petri dish in the operating room to learn that there are plenty of organisms in the air and some must occasionally enter the wound. Our aseptic efforts, therefore, reduce infection to a minimum rather than completely prevent it. The second difference is that the tissues which are closed over the bone are normal in their blood supply and are undamaged by any serious trauma. If by treatment we could transform a compound fracture into something which resembled an operative wound we might hope for control of the infection and primary healing of the wound. This is the rationale of debridement.

Principles of Treatment

1. *Debridement.* Debridement is by far the most important measure in the treatment of compound fractures. It

is a rational attempt to convert a traumatized and infected wound into a clean wound lined with healthy tissues. The outcome of our treatment of compound fractures depends more upon the manner in which the debridement is carried out than upon any other phase of the treatment. The ideal at which we aim is the complete removal of all bacteria which are in the wound and the complete removal of all dead and devitalized tissue. Under ideal circumstances both these ends can be achieved. Under less favourable circumstances they can be approximated and the healthy tissues lining the wound can be expected to dispose of a very considerable number of the bacteria which may remain.

2. *Technique of Debridement.* How should this most important principle of debridement be carried out? First, it must be done as quickly as possible after the injury. At the inception of the wound, the bacteria are superficially placed; rubbed into the surfaces of the wound by contact with clothing or earth or pavement. As time passes they establish themselves in the tissues and grow, invading their surroundings. For a few hours after the injury their mechanical removal is easy. After that it is difficult or impossible.

The limb surrounding the wound of the compound fracture must be made surgically clean. Nearly all compound fractures are sustained under circumstances which make the surroundings of the wound dirty and infected. It is necessary that the region of the wound be surgically clean before the actual debridement is commenced. Since soap and antiseptics are powerful irritants of tissues other than the skin the wound must be closed or covered while the limb is cleansed and prepared. It is best to close the wound snugly with sutures where this is possible. The limb is then prepared as for any operation, first by mechanical cleansing and secondly the use of skin antiseptics. The cleansing is the more important. The part must be made surgically clean and sufficient time and sufficient mechanical scrubbing with soap must

be utilized to achieve this end. When the limb is thoroughly clean and dry, the gloves, linen and instruments are changed, iodine is applied and the wound reopened by removal of the sutures.

The debridement itself consists in (1) excision of the lacerated skin margins; (2) removal of all damaged tissue except nerves, vessels and intact bone; (3) removal of all dirt and foreign bodies; (4) perfect haemostasis. After the sutures which close the wound have been removed, the lacerated skin margins are excised back to healthy skin. No more skin than is absolutely necessary should be removed. The skin wound then should be enlarged freely in order to permit adequate exposure of all the depths of the wound. The damaged soft tissues are removed freely by scalpel and scissors until only healthy tissue remains, tissue which bleeds freely and if muscle, contracts when pinched. Particles of dirt and clothing must be carefully searched for and removed. Mechanical scrubbing by a soft brush or a piece of gauze supplemented by abundant and prolonged irrigation with sterile saline is of great value at this stage. When all the devitalized tissue has been cut away, such mechanical flushing and rubbing (though not so vigorous as to be traumatizing) will most effectively remove the particles of dirt and bacteria. The fractured bone ends can be scrubbed clean with a brush. They should not be resected and no bone should be removed unless it is completely detached from its blood supply. It goes without saying that vital structures such as nerves and blood vessels are not included in the excision. They must be preserved for future function and vitality of the limb. Cleansing of them must be limited to wiping them with gauze under the flushing of a stream of saline. When the wound is clean and free of dirt, careful haemostasis is performed. There must be no oozing to permit the accumulation of blood clots. The sooner after injury the debridement is commenced, the more meticulous the care, the more painstaking the cleansing of every nook and cranny of the wound,

the more abundant the flushing with saline, and the more perfect the haemostasis, the more nearly will the debrided wound approach the ideal of the operative wound.

There is to-day little difference of opinion regarding the merits of debridement. All agree that it is *the* important first step in the treatment of compound fractures. There may be some difference of opinion as to how far debridement should go. I have indicated that I think it should be early, extensive, prolonged and thorough. If we bear in mind what we are struggling to achieve by debridement (convert the compound fracture into a simple fracture) there can be little doubt as to the pains which must be taken in its performance.

3. *Treatment After Debridement.* Debridement completed, we are faced with the problem of what to do next with the wound and here there is some difference of opinion. There are at least three schools of thought. *First*, immediate closure in an attempt to achieve primary healing. *Second*, the use of antiseptics followed in a short time by delayed primary suture. The antiseptic may be Dakin's solution instilled by the Carrell-Dakin technique or it may be Rutherford Morrison's B.I.P.P. *Third*, the vaseline pack and closed plaster treatment of Orr and Trueta.

Orr's long advocacy of the vaseline pack and closed plaster and Trueta's experience with this method in the Spanish War, together with his startling results (eleven hundred cases with only six deaths), has aroused an immense enthusiasm for the method. There can be little doubt that it represents an important advance in the treatment of compound fractures due to war wounds. The great extent of war wounds, the extreme degree of pulping of the tissues, the number of wounded who must be treated and the speed with which operations must be performed all tend to result in imperfect debridement with a considerable degree of residual infection in the wound. Under such circumstances primary or delayed primary

closure cannot be performed. Some form of drainage must be established and the vaseline pack plus closed plaster has merits above all others. It enables large numbers of war casualties to be dealt with quickly and adequately; the proof of this is the low mortality and the low incidence of gas gangrene and amputations. The wound packed open with gauze is virtually exteriorized and ready drainage occurs between the vaseline of the pack and the wall of the wound (curtain drainage). The plaster fixation is important; it keeps the tissues at rest and this facilitates the building of tissue barriers against infection; it facilitates early transport; the elimination of dressings is a comfort to the patient and a relief to the overburdened staff of a war hospital, as well as a protection against the implantation of haemolytic streptococci in the wound. Simplicity, speed, ease of transport, freedom from dressing, but above all the high recovery rate and the low incidence of gas gangrene and streptococcic infections are the merits of the Orr-Trueta treatment which make it a revolutionary advance in war surgery. It cannot, however, be advocated as the ideal method of dealing with fractures. The reduction and fixation of the fractures in the plaster often is imperfect. There is an unnecessary amount of joint stiffness. Wound healing is by granulation with the formation of much scar.

In spite of its merits as a war measure, it is not by any means certain that the Orr-Trueta closed plaster treatment is the ideal method for use in civil and industrial surgery. A considerable proportion of such cases are of such a nature that effective debridement can be followed by primary suture with the expectation of primary healing. In civil and industrial surgery, the ideal at which we should aim is such early and effective debridement that primary suture may be carried out. Civil surgery is operating under ideal conditions, not pressed for time. A great many compound fractures in civil life are not badly traumatized as are war wounds. A proportion of our compound fractures

should, therefore, be capable of primary closure. The forms of open treatment—Carrel-Dakin, Morrison's B.I.P.P., and the Orr-Trueta method—should be reserved for the few worst problems. This point of view needs emphasis at the present time since there is a strong tendency to translate the enthusiasm for the Orr-Trueta method in war wounds to the problems of civil life where it is largely unnecessary. To advocate strongly the Orr-Trueta method and chemotherapy for the treatment of compound fractures, tends to detract attention from the importance of debridement. This would be a serious mistake. Nothing we now utilize in the treatment of compound fractures is of as great value as debridement. The vaseline pack and plaster are an acknowledgment that for some reason debridement has been imperfect. It is a method of open drainage; one which has considerable merit, but needless if we can control the infection by debridement or by debridement supplemented by sulphathiozole.

4. *Chemotherapy.* No discussion of to-day's treatment of compound fractures can overlook the part which may be played by sulphonamide drugs in the management of the infection. Sufficient time has not yet elapsed to permit us to pass final judgment upon their value, or upon the best type of sulphonamide to use, or upon the best method of administration. But all the evidence which accumulates points with increasing force to the important part they can play. There is much evidence to make us believe that the sulphonamides give the final touch to the debridement and dispose of the last few bacteria in the wound. From now on the sulphonamides are likely to become an integral part of the treatment of compound fractures. The impressive results obtained by the sulphonamides in the treatment of osteomyelitis must surely indicate that the sepsis of compound fractures can be equally well controlled.

Such experience as has accumulated indicates that sulphathiozole is the most useful drug and that it should be

administered both locally into the wound and also by mouth. It is important to saturate the patient and the wound quickly with the drug.

5. *Anaerobic Infections.* Though anaerobic infections are uncommon in compound fractures, when they do occur they are of such seriousness that every care should be taken to avoid them. The prophylactic measures of value are, (1) The removal of every particle of dead tissue from the wound. They will establish themselves in the wound only if dead tissue is present. (2) A painstakingly careful debridement of the wound with prolonged flushing with sterile water or saline. (3) The administration of antitetanus serum and antigas gangrene serum to all compound fractures. The development of an active immunity against tetanus by means of tetanus toxoid is important but probably we can never attain this on a wide scale except in such organized groups as the Army. The best measure is prevention and the best prevention is debridement.

6. *Haemolytic Streptococcic Infection.* In the war of 1914-1918, the commonest infecting organism in compound fractures was the haemolytic streptococcus and it also was the most serious. To a considerable degree the same statement holds true for the compound fractures of civil life. The method by which a wound becomes infected with haemolytic streptococcus is somewhat unusual and deserves a moment of our attention. The work of Ronald Hare of the School of Hygiene has shown that the great reservoir of haemolytic streptococci is the nasopharynx of human beings. They are not present on street pavement, machinery or soil. They can exist only in the human nasopharynx or in wounds. Compound fractures which become infected with haemolytic streptococci do so because the organisms are implanted in them directly from the mouth secretions of attendants or indirectly from hands, instruments or bed clothes. They do not enter the wound at the time of the accident but for the most part are secondary invaders breathed or coughed or talked into the wound while it is being examined or

dressed. One of the great merits of the Orr-Trueta method is the exclusion of the haemolytic streptococci by the unopened plaster, a feature clearly recognized by Orr himself who repeatedly urged its use on the grounds that it prevented secondary streptococcic infection. Our whole knowledge of this organism, its source, its manner of entering wounds, its seriousness and its great powers of invading tissue is a strong argument for the infrequent dressing of wounds. This in turn is an argument for primary closure of the wound.

7. *Plastic Procedures to Secure Primary Closure.* If there has been considerable loss of skin it may be difficult or impossible to secure primary suture of the wound. In such cases skin plastics can be used to permit the wound edges to be sutured without tension. It is necessary to cover the fracture with an adequate layer of soft tissues if the best result is to be obtained. Plastic procedures should be used more often for this purpose. Cushing's work on cranio-cerebral injuries in the last war demonstrated that primary suture after debridement was the best method of treating such wounds. Often to obtain primary closure skin plastics were necessary and he strongly advocated their use. The same advantages occur in the management of compound fractures of the extremities. Some standard plastic procedures which are of value in obtaining this result are (1) relaxation incisions to permit suture of the wound margins without tension. The areas denuded by this skin sliding are immediately covered with a Thiersch graft. (2) Lateral curved elongations of the wound to permit sliding of the skin margins and suture under less tension. (3) The complete removal of areas of undermined skin and its replacement as a full thickness graft after removal of the subcutaneous fat. This is A. W. Farmer's procedure and is a most valuable adjunct to the management of badly lacerated wounds.

8. *Management of the Fracture.* Compound fractures have one advantage over simple fractures. The fracture site

usually can be seen clearly and perfect reduction of the fracture can be obtained by direct manipulation of the fragments. Often this is of great value, especially when the fragmented ends are so shaped as to permit the reduction to be retained once it has been achieved. In the management of the more difficult fractures, I feel certain that the more frequent use of internal fixation or skeletal transfixion are justifiable. As we improve our methods of handling infection by better debridement and by chemotherapy, the wound so much the more closely resembles an open operation on bone, which invites and justifies the use of methods of internal fixation. It has been shown by Sherman and others that internal fixation by screw and by plate and screw often is good treatment even if the plate must later be removed. The accuracy of reduction so attained and maintained and the improved prospects of rapid union with minimum residual disability more than compensate for the need of a second operation. The Kirschner wire also provides us with a simple means of transfixing fragments and incorporating them in plaster. In properly selected cases simple forms of internal fixation have much to recommend them.

Summary

The treatment of compound fractures to-day is a problem of great importance because of the steadily increasing numbers of injuries in industry and in motor traffic. Infection is the problem of

outstanding importance. The principles of treatment established in the last war still hold and they have been supplemented by new chemical agents of great efficiency. Our aim should be to convert the infected wound into one such as might have been produced by a clean surgical operation. For the accomplishment of this, debridement is the procedure of greatest value. It will succeed (1) if it is done early; (2) if all dead and devitalized tissue is removed; (3) cleansing should be mechanical rather than chemical. Prolonged irrigation with sterile water or saline is valuable. The aim should be to cleanse the wound of all infections so that it can be closed by primary suture with the expectation that it will heal. (4) Sulphathiozole in the wound and by mouth is a valuable adjunct of treatment. (5) The Orr-Trueta method should be reserved for cases in which adequate debridement cannot be carried out. It is a method of drainage for established infection. It is not a good method of treating the fracture unless supplemented by traction or by transfixing pins, and even then it results too often in stiff joints. (6) Skin closure should be attained even if skin plastics are necessary for this. In appropriate cases (fractures difficult to retain, good reason to believe that debridement is successful) internal fixation even by plate and screw is justifiable and wise. (7) Improved management of infection justifies the use of internal fixation, especially since we now have simple atraumatic apparatus for the purpose.

THE TREATMENT OF TRICHOMONAS VAGINALIS INFECTION

By JAMES C. GOODWIN, M.D.

*Junior Demonstrator in Obstetrics and Gynaecology,
University of Toronto*

Despite the fact that the primary purpose of this paper is the discussion of various aspects of the treatment of *Trichomonas vaginalis* infection, a consideration of some of the clinical features and related laboratory investigations and findings may not be out of place. In the recent literature, increasing prominence has been given to this interesting and rather curious protozoan—the *Trichomonas vaginalis*, whose ubiquity is becoming more widely recognized; and whose activity in the production of distressing symptoms is bringing, and will continue to bring many women for relief, to the door of the general practitioner or gynaecologist.

Though Donné (1) originally discovered the *Trichomonas vaginalis* apparently he was not convinced that the protozoan represented a very likely cause of the vaginal discharge in which he found it. Approximately twenty years later in 1855 Koelliker (2) confirmed this observation, and as far as is known, was the first to note a relationship between this motile flagellate and an irritating vaginal discharge—and also the absence of the trichomonads in the normal vagina. From then until the past decade, the literature has contained much for and against the view that the abnormal vaginal discharge was the result of *Trichomonas vaginalis* infection. Recently, however, it would appear to be well established that the *Trichomonas vaginalis* exists specifically as the causative factor in the production of a definite clinical entity. Common clinical observation will establish the relationship between the protozoan and a typical vaginitis, which disappears with the disappearance of the flagellates; recurring if the latter are not completely eradicated; with the patient remaining cured if no further trichomonads can be found. The problems which now con-

front the observer concern its source, the mode of access of the organism to the vagina and other features of its life history; as well as the method by which one can eradicate it from the human soil in which at times it seems so deeply implanted.

The prevalence of *Trichomonas vaginalis* infection has been the subject of comment by many writers, (3, 4, 5, 6, 7). Reports indicate a world-wide distribution in civilized areas. Whereas some statements suggest a very high incidence of this infection, one is of the opinion that only about 10 per cent of gynaecological clinic patients present evidence of the *Trichomonas* infection of vagina. In a more limited group of patients, however, coming for the treatment of persistently abnormal vaginal discharge, a much higher percentage of *Trichomonas* vaginitis cases may be expected—25-40 per cent. In venereal disease clinics this is particularly true. Here one observes gonorrhoeal patients apparently failing to respond to treatment, the continuing discharge subsequently being found to be due to *Trichomonas vaginalis* infection. Prenatal clinic patients do not present the infection quite as frequently as do non-pregnant patients.

Clinically one finds an acute, a sub-acute and a chronic form of *Trichomonas vaginalis* infection involving the lower genital tract. The sub-acute and chronic forms may be simply later stages of the acute infection, or either may appear as the initial picture. The acute form of *Trichomonas* vaginitis is mistaken not infrequently for active gonorrhoea. This necessitates a careful diagnosis in order to prevent unwarranted accusations of marital infidelity. The patient acutely infected complains of a profuse irritating vaginal discharge, intense smarting on voiding, soreness on walking in the vulvar

ness, emotional irritability and mild fatigue, — common associated general symptoms. Examination reveals a copious thin creamy vaginal discharge, yellowish white or a faint greenish yellow in colour. It is not always frothy in character. The discharge has a sourish rancid somewhat musty unpleasant odour which is characteristic,—and yet defies accurate description. Once associated with the clinical picture it is rarely forgotten. Varying amounts of labial oedema are evident with patchy reddening of the vulva and inner aspects of the thighs. Marked tenderness is found. This makes pelvic examination very trying to the patient,—and demands the utmost gentleness on the part of the physician. In the acute form, one finds reddening of the vestibule and urethral orifice, but no urethral discharge, thus differing from acute gonorrhoea in this particular. The vaginal walls appear roughened, present marked reddening, especially in the vault, where characteristic punctate hyperemic petechia-like areas may be observed. These are more common in the mucosal surface of the cervix and vaginal vault, than in the lower vagina.

The sub-acute form may follow the acute, or as stated may represent an initial process, due to either a less active trichomonad, or what is more probable — *a less favourable environment for growth*. Here one observes a less diffuse reaction in a lesser degree. The inflammatory changes in the vagina are most marked in the vault. General symptoms tend to be slight or absent, though local symptoms are aggravated by menstruation or coitus. In the acute form, coitus may be very distasteful to either partner because of the unpleasant vaginal discharge, and the degree of dyspareunia depends on the intensity of the inflammation.

The chronic or latent form may represent a later phase of an acute or sub-acute process, or again *dependent on an unfavourable habitat*, the initial foothold gained by the flagellate may be slight. In fact a casual examination may fail to reveal the condition. A scanty thin yet abnormal discharge is present, with slight

but usually characteristic evidence in posterior aspect of the vaginal vault. The patient exhibiting this form of *Trichomonas vaginalis* infection may complain of little other than a slight irritating discharge and dyspareunia, with discomfort aggravated by menstruation, coitus and warm weather.

In cases of post-operative, post-abortive or post-partum infection coincident with *Trichomonas vaginitis*, fever is due rather to associated pathogenic bacteria than to the protozoa. Three patients have been seen recently in which acute *Trichomonas vaginitis* existed in the first trimester of pregnancy, even going into labor without entire freedom from abnormal discharge, and yet subsequently proceeding throughout the puerperium without febrile disturbance.

Diagnosis is not difficult—only the chronic or latent form requiring other than ordinary care to recognize the cause. Where the patient has taken a douche within twenty-four hours, or where an antiseptic lubricating jelly has been used on the examining glove or speculum, one may fail to discover the flagellate due either to reduction in number, or to temporarily lessened or absent motility. Clinically, if one has reason to suspect *Trichomonas vaginalis* as the cause, it is wiser to defer search for the protozoan until another twenty-four hours have elapsed without douching. Where possible, vaginal discharge from the region of the vaginal vault should be obtained for diagnosis. For this one may use either a clean pipette or a cottoned applicator to convey the discharge to 4-5 cc. of normal saline, or better still Locke's solution. Either diluent for the discharge is best kept at body temperature in a water bath, as this temperature maintains the optimum motility of the flagellate. After mixing the discharge well in the diluent, a drop or so of this is transferred to a warm, clean slide and covered with a cover-slip to prevent evaporation. Under the L.P. or H.P. (Dry) lens the protozoa may be seen wriggling in and about the cellular debris, epithelial and pus cells characteristic of the discharge. The *Trichomonas vaginalis* is about 16-28

microns long, "snowshoe" shaped, having four anterior flagella, and a posterior tail-like axostyle. The movement of the flagella axostyle and an antero-posterior undulating membrane serves to give it a typical "jiggling" movement. The protozoan is larger than a pus cell and smaller than an epithelial cell. The observation of other physical characteristics depend upon certain staining methods—which procedure is not necessary for diagnosis.

Differential Diagnosis must exclude gonorrhoea, monilia albicans, thrush and other yeast forms, as well as vaginitis due to other bacterial organisms such as streptococcus, staphylococcus and the colon bacillus, etc.—related to local or pelvic inflammatory pictures. The methods required to establish diagnosis of other causes of infection than *Trichomonas vaginalis* are in the main simple and need not be outlined here.

Recent investigations in this field, have directed considerable interest to the vaginal pH factor in relation to *Trichomonas vaginalis* infection. Observers (6), (8), (9), (12) have found the normal pH of the vagina to lie between 2.5 and 4.5. Trussell (8) points out that normally a lower pH exists nearer the vaginal outlet than at the vault. In *Trichomonas vaginalis* infections, the pH of the vagina tends to rise to 5-7. Liston and Liston (11) believe "that the pH of the vaginal secretion plays an important role in bringing about primary infection by the *Trichomonas vaginalis* and in maintaining it once infection has begun". He has found that the pH must be maintained around a point slightly on the alkaline side of pH 6 for the successful development and multiplication of the *Trichomonas vaginalis*. A more alkaline pH permits growth of pathogenic bacteria as well, which further adds to the inflammatory picture. This association of the *Trichomonas vaginalis* with certain bacterial organisms not normally found in the vagina has been regarded by some as a symbiosis. Such organisms as the streptococcus sub-acidus, alpha streptococcus viridans, gamma and beta haemolytic streptococci are reported (12).

Greenhill (12a) in the same connection points out that it is being recognized more and more that the reaction of the vagina plays an important part in the successful treatment of vaginal discharges, as it would appear that no pathogenic organisms can survive indefinitely in a medium below a pH of 5. It is known that the pH of the vagina swings to the alkaline side during and immediately following menstruation, and after repeated coitus. This is true also during the development of inflammatory states, and after douching with strong antiseptics. Here exudations occur in the vagina which alter the pH toward a lessened acidity. It is a matter of common clinical observation that the onset and exacerbation of *Trichomonas vaginalis* infection tends to follow menstruation and repeated coitus. As pointed out above this infection once begun tends to keep the pH at a level suitable for the continued multiplication of the flagellates by the production of an inflammatory vaginal reaction with subsequent exudation. It is logical to infer from these observations that any treatment which aids in the restoration of the normally acid pH of the vagina, *produces a habitat unfavourable to the Trichomonas vaginalis* and hence favours a cure. This has been borne out by clinical experience.

Origin of *Trichomonas Vaginalis* Infection

The query as to the origin of *Trichomonas vaginalis* still remains unanswered. Certain workers (13), (14), (14a) using various cultural and staining methods claim the *Trichomonas vaginalis* to be distinctly an individual type, and not related to other human types. On the other hand, opinions (6), (15), (16), (17) have been expressed that they are all identical. Wenyon (18) a competent authority on protozoa believes that it is quite possible that the three types of human trichomonas, *T. vaginalis*, *T. buccalis*, *T. hominis* belong to one species and that the differences which occur are due to variations in nutrition and environment. Karnaky (6) inoculated eight women known to be free from *Trichomonas vaginalis* infection with an intes-

tinal trichomonad. Three of the eight acquired infection, the intestinal trichomonad changing over to the vaginal type in 4-5 days. Zener (19) recently reported four *Trichomonas vaginitis* cases in which identical trichomonads were recovered from both vagina and rectum. In two of these cases the vaginal infection followed attacks of diarrhoea. Peterson (20) reported a case of acute proctitis complicating *Trichomonas vaginitis*; and in both areas the trichomonads were found to be identical. The transmission of the protozoan from bowel to vagina in a medium favourable for its growth—alkaline menstrual fluid—would appear very probable in the light of foregoing observations. One is strongly of the opinion, therefore, that certain morphological and even cultural characteristics in as primitive a type of life as the *Trichomonas vaginalis* may be acquired; and that with better methods of recovery of the trichomonads from the stool or mucosal surface of the rectum, the intestinal origin of this vaginal infection will yet be proven and accepted. Thus intestinal as well as vaginal treatment will be necessary to prevent recurrence due to reinfections. Other opinions as to the origin of the initial infection have been given—due to contaminated bathing pools and bathtubs, toilet articles, douche equipment and faulty douching, wearing of recently used underclothes, etc.

Recurrent Vaginal Infection

The likelihood of recurrent vaginal infection from the spread of trichomonads from intestine has been discussed. One case in the series reported below presented a chronic form of *Trichomonas vaginitis*—the attenuation of an acute picture some six months before. Irritation was occasionally noticed within the anal canal and this suggested a probable explanation for periodic recurrence in spite of long continued vaginal therapy. Proctoscopic examination revealed a slightly inflamed anorectal mucosa with sufficient exudate to recover a trichomonad identical with that observed vaginally. Permanent freedom from periodic recurrence in this case still eludes treatment, in spite of the

absence of other foci of *Trichomonas vaginalis* infection and abstinence from intercourse. A very likely source of reinfection in *Trichomonas vaginitis* exists in the male, according to Riba (21), Karnaky (6), Moench (22), Drummond (23) and others. Here the husband becoming originally infected from his wife, serves to reinfect her periodically—making a cure almost impossible until the source is recognized. These observers have found *Trichomonas vaginalis* infection in a fairly large number of males, foci being located in the urethra, the prostate or under the prepuce—sites commonly having an alkaline pH. Apart from mild itching or slight whitish morning discharge these men may have no symptoms.

Other sources of reinfection are claimed for the bladder (12). In this connection the *Bodo urinalis*, another protozoan form, in all probability frequently has been mistaken for the *Trichomonas vaginalis*. Foci have been reported to exist in Skene's tubules and Bartholian's glands (24). Chronic infection of the lower cervical canal seems to represent a likely focus with outpouring of alkaline mucus; though little evidence has been adduced in favour of the uterus, tubes, or pelvic peritoneum as possible foci as stated by Goodall (25). Pattyson (12) in this connection, found no *Trichomonas vaginalis* in the examination of the endocervix, uterine cavity or kidney pelvis in 250 cases.

Treatment

Notwithstanding the numerous papers in the past few years dealing with the treatment of *Trichomonas vaginalis* infection, no specific yet exists. Practically any chemical will kill the *T. vaginalis* in vitro. Almost every known antiseptic has been tried—wet and dry treatments—douches, applications, ointments, pastes, tampons, tablets and powders; substances taken by mouth and by hypodermic, substances to lower the vaginal pH; protozoacidal substances, etc.—with varying results: some very useful, others fair, many useless and some actually harmful. Space prevents more than mere mention

of some of them, comment being reserved for those found to have the most practical value.

Innumerable varieties of cleansing douches have been recommended; some of them possessing sufficiently strong chemical qualities as to injure the vaginal mucosa, causing exudation and lowering tissue resistance to other organisms. Especially is this true where vigorous "scrubbing" of the vagina has preceded their use. Patients themselves have been known to produce harmful results with strong solutions of lysol and other strong antiseptics. Mercuric bichloride solution has been used as a douche and on tampon (22)—not without danger of absorption; mercury oxycyanide 1:4000, merthiolate 1:1000, potassium permanganate 1:4000 have been tried (12) with unsatisfactory results. Alkaline substances such as sodium bicarbonate, sodium perborate, magnesium sulphate, borax, etc., are useless because of the effect of the pH on the vagina.

Arsenical substances given orally and intramuscularly have been found to be of little appreciable value—and the same applies to intramuscular bismuth. The arsenical substances are not without possible toxic effects, some of which may be severe. Local treatment in tablet or powder form with such arsenical compounds as aldarson, stovarsol, acetarsone, devegan, carbarsone, etc., have proven in the main useful, though toxic effects may occur from vaginal absorption because of the relatively large dose used (27). Their usefulness in some instances is definitely limited unless the treatment includes some substance to assist in restoring the normally acid pH of the vagina. Pattyson (12) reports excellent results using locally a pentavalent arsenical, cinquarson (grs. 15) diluted in boric acid, powdered for vaginal insufflation. In addition, for recurrent cases, a bladder instillation of the same substance ($7\frac{1}{2}$ grs. in 50 c.c.) in aqueous solution was administered three times in two weeks. He claims 96 per cent of cures with the combined vaginal and bladder method. Zener (19) believing in the rectal origin of *T. vaginitis* reports the use of another protozoacide, iodo-chlor-oxyquinoline in over 200 cases.

The drug was diluted in 1 part with 9 parts of magnesium trisilicate; 50 grains being used locally in powdered form for insufflation three times a week for one complete menstrual cycle; with 4 grains of the drug by mouth t.i.d. for ten days. He claims over 95 per cent cures; the cure being the absence of the *Trichomonas vaginalis* on bi-weekly examination for three months after treatment is concluded. Cherry (26) claims excellent results using estrogenic hormone intramuscularly in conjunction with a mandelic acid buffer (having a pH of 3.0) insufflated vaginally—which consists of 4.29 grams sodium mandelate, 3.16 grams mandelic acid and 1.0 grams glucose.

In order to evaluate the usefulness of silver picrate in the treatment of *Trichomonas vaginalis* infection, a series of 100 consecutive cases is reported in which this substance was used routinely. This series consists of out-patients coming to the Toronto General Hospital Gynaecological Clinic. In each case the diagnosis of *Trichomonas vaginalis* was based on the finding of the *T. vaginalis* in the vaginal discharge, in addition to the clinical findings. Cases were subdivided into 27 acute, 24 sub-acute and 49 chronic. The entire group was divisible into 72 married and 28 single patients. Approximately 75 per cent of the patients were between 20-39 years of age—the youngest 16 and the oldest 59. There were 44 nulliparas, 14 primiparas, and 42 having two or more children. 16 patients in the series had gonorrhoea as well as *Trichomonas vaginalis* infection, with positive cultures for the gonococcus; 7 had syphilis with positive Wasserman reactions. Of these 23 venereal cases, three had both gonorrhoea and syphilis as well as the *Trichomonas vaginalis* infection.

All patients were placed on the following routine of treatment—commencing where possible at least two weeks before the next period was due:

1. (a) A insufflation of 5 grams of silver picrate (1% in powdered Kaolin) with a rubber guard type of insufflator—allowing sufficient pressure to disperse the powder into the air-dilated vaginal canal.

- (b) Patient given 6 vaginal suppositories, each containing 2 grams of silver picrate in a boroglyceride base—with instructions to insert one each night for six nights and then to return to clinic in one week.
 - (c) Patient further instructed to take only sponge baths during treatment and to avoid douching or coitus until cured.
2. One week later:
- (a) Insufflation repeated.
 - (b) Further course of six vaginal suppositories as above.
3. Following next menstrual period:
- (a) Smear taken for diagnosis.
 - (b) If positive repeat routine.
 - (c) If negative patient instructed to return following next menstrual period for re-check and again in 3-6 months for final re-check. Where recurrence is found routine is begun again.

For the purpose of this study I have listed as:

Cured patients whose smears were negative following two menstrual periods.

Doubtful cures—those whose smears were negative following one menstrual period, and

Improved—those in whom the *T. vaginalis* could not be found—but whose discharge and other clinical evidence was not entirely eliminated.

Recurrences were listed as those in whom the smear showed *Trichomonas* after being negative following the first menstrual period.

Not improved included those in whom the *Trichomonas vaginalis* persisted along with other clinical evidence on examination.

The usual problem of obtaining follow-up visits existed with this group. Notwithstanding an excellent social-service department, an adequate follow-up was exceedingly difficult to secure in the majority of cases. Allowing the treatment full benefit of any doubt, I have included for the purpose of summarizing

results: *cures*, *doubtful cures*, and *cases improved*, in one group; and then subtracting the known recurrences, have termed this balance *possible cures*.

In this series the following results were obtained:

- (a) acute cases —possible cures—78%
- (b) subacute cases—possible cures—71%
- (c) chronic cases —possible cures—61%
or 68% for the whole group.

Failure to respond to treatment:

- (a) acute cases —11%
- (b) subacute cases—25%
- (c) chronic cases —24%
or 21% for the whole group.

In addition to:

11% of cases of known recurrence following treatment.

There were 3 cases which displayed sensitivity to silver picrate following one week of treatment, necessitating discontinuance of the method. In many cases, however, treatment was continued for much longer than the routine two weeks in the hope of obtaining a cure, and before concluding failure of treatment.

The results in this series of cases treated with silver picrate according to the method outlined—(admittedly containing considerable likelihood of error in evaluation of *possible cures*—certainly fell far below the results claimed by Buxton and Shelanski (7). These observers claim in 100 cases—100% disappearance of the trichomonads and with the disappearance of symptoms including discharge and irritation in over 90 per cent—following two weeks of treatment on the same routine as outlined above. They reported the return of positive smears in only two cases (18 and 21 weeks after treatment respectively) and believed these to be due to actual reinfection rather than to recurrence. 62 of their cases were followed for six months and 38 for nine months—a remarkably complete follow-up service record.

One finds it very difficult to account for such a discrepancy in results—especially when care has been taken to ensure adequate, and in many cases more than what has been considered adequate treat-

ment. I believe, however, that silver picrate treatment for two weeks, as outlined above, possesses great merit in relieving the symptoms of discharge and irritation. Especially is thus true in the acute form of *Trichomonas vaginitis*—comfort being restored in the majority of cases well within the two week period. Nevertheless, on the basis of the results reported above, and from a general knowledge of the infection, I am very dubious as to the ability of this or any other method of therapy so far available to effect, within two weeks, a rapid or permanent cure in 100 per cent of cases of *Trichomonas vaginalis*. Recurrence is not necessarily always the fault of the vaginal treatment—as it may be due to incomplete pelvic eradication of the *Trichomonas vaginalis* or to vaginal reinfection.

Until some specific cure is obtained, and definite knowledge exists as to the origin of the initial infection, and furthermore until the causes of recurrences or reinfection are fully understood, treatment based on pH studies, appears to possess the greatest practical value. In the meantime the best method of obtaining rapid symptomatic relief in *Trichomonas vaginalis* in my opinion is the repeated vaginal insufflation of 1 per cent silver picrate in kaolin and in the coincident use of the same drug in the form of vaginal suppositories—according to the method previously outlined. This drug has the further advantage of quickly eliminating associated inflammation due to pathogenic bacteria. Once the comfort of the patient is obtained, it can be maintained by restoring an acid pH in the vagina by the use of simple substances in douche form such as lactic acid (1 dram to the quart) or vinegar (5-6 tbsps. to quart). These possess a known low pH, the effect of which is to restore and maintain a more normal vaginal pH, which in turn induces the return of the normal anti-pathogenic vaginal bacterial flora, and the disappearance of the trichomonads.

Following the restoration of the patient's comfort by the method sug-

gested, a daily douche throughout one complete menstrual cycle should be used excluding only the heavier days of the menstrual flow—followed by douches on alternate days during the second menstrual cycle. Then one or two douches a week are advisable for a sufficient time (3-6 months) to eliminate the possibility of recurrence. Where the patient has remained well the low pH douches may be discontinued if a few mid-cycle examinations (nitrazine paper) show a persistently normal vaginal pH and the absence of *Trichomonas vaginalis*. This plan of treatment causes the patient very little inconvenience, is inexpensive and can be quite safely recommended.

Conclusions

1. It would appear well established that *Trichomonas vaginalis vaginitis* is a specific disease; being caused by a motile unicellular flagellate protozoan.

2. *Trichomonas vaginalis* infection is common—occurring in approximately 10 per cent of gynaecological clinic patients; and in 25-40 per cent of patients with gonorrhoeal infection.

3. Clinically one observes an acute, a subacute, and a chronic or latent form of *Trichomonas vaginalis vaginitis*, each presenting a more or less characteristic picture.

4. The disease bears considerable resemblance chemically to gonorrhoea but may be readily distinguished from it.

5. Diagnosis is simple—being based upon the microscopic recognition of the motile protozoan parasite in a drop or two of a saline suspension.

6. The pH of the vaginal discharge in *Trichomonas vaginalis vaginitis* usually ranges between 5-7; that of the normal healthy vagina being 3.5-4.5.

7. Evidence would suggest a rectal origin of the infection—and that the oral, intestinal, and vaginal types of *Trichomonads* belong to one species—differences being due to variation in nutrition and environment.

8. Reinfection can occur from a male previously infected at intercourse,—the trichomonads being harbored in urethra, prostate and beneath the prepuce.

9. Various treatments and their merits are briefly discussed, including a report of 100 cases treated with silver picrate. The latter treatment possesses the greatest value in the acute form of *Trichomonas vaginalis vaginitis*.

10. A suggestion is made as to an adequate, simple routine of treatment

pending the discovery of a specific cure for *Trichomonas vaginalis vaginitis*.

I would like to express my appreciation to Professor W. A. Scott of the Department of Obstetrics and Gynaecology for his permission to report the series of cases included in this paper, to Dr. George Hendry for his very kind assistance with the analysis of cases reported, and to Mr. Lee of John Wyeth & Brother Company for the generous supply of silver picrate used in the treatment of these patients.

Bibliography and reprints may be obtained from the author on request.

THE RH FACTOR AND BLOOD TRANSFUSION

By SAMUEL HANSON, M.D.
Toronto General Hospital

The recent discovery by Landsteiner and his co-workers of another agglutinin in human blood makes necessary a reconsideration of our criteria of compatibility of blood for transfusion. This new agglutinin is the explanation of most cases of haemolytic reactions which occur even though the recipient and donor belong to the same blood group.

As well as two agglutinogens A and B, which divide human bloods into the classical four groups O, A, B and AB, additional agglutinogens M and N were described by Landsteiner and Levine fifteen years ago. These agglutinogens divide all human bloods into three further types—M alone, N alone and both M and N. No blood has been discovered which lacks both these factors, i.e., there is no minus M minus N blood. Shortly thereafter the same investigators reported another agglutinin in human red cells which has been named P. In 1940 still another agglutinin was discovered by Landsteiner and Wiener. They designated this factor Rh (rhesus-human factor) because it appears to be identical with an agglutinin present in rhesus monkey blood. The agglutinin Rh has been quite extensively investigated by its discoverers and their work shows that this property is involved in most of the cases of intra-group transfusion reactions.

The Rh factor is present in 85 per cent of human bloods. The remaining 15 per cent lack it.

If an Rh positive blood is injected into an Rh negative individual anti-Rh agglutinins may be formed. This may occur as early as twelve days after a transfusion of Rh positive blood. A subsequent transfusion of Rh positive blood will then result in a haemolytic reaction. We see, therefore, that in these cases the recipient must always be Rh negative and the offending donor Rh positive.

But a haemolytic reaction due to Rh factor may also occur following the first blood transfusion. Here the explanation

is somewhat more involved. It hinges on the fact that in all cases of haemolytic reactions following a single transfusion reported in the literature the recipient has been a post or intrapartum woman. Most of these cases are now believed to be due to an anti-Rh agglutinin. This agglutinin was formed not in response to a transfusion of Rh positive blood but to an Rh positive foetus in utero; the foetus having inherited the Rh factor from an Rh positive father.

Wiener (2, 3) has reported 15 cases of intra-group incompatibility due to Rh factor, four of these were fatal. In each case the recipient was found to be Rh negative and either had received more than one transfusion or was post partum. In several of these cases subsequent transfusions were uneventful when known Rh negative donors were used. This investigator recommends that post partum women be transfused only with Rh negative blood.

Technique of testing for Rh agglutinin.

To detect the Rh agglutinin it is necessary to carry out a tube test at ice-water temperature. The following is the technique Wiener and Peters described in 1940. To two drops of recipient's serum in a Widal tube is added one drop of donor's cell suspension. A control is always used of recipient's serum and recipient's cells. The two tubes are immersed in ice-water for five minutes and then centrifuged for two minutes. A positive reaction is recognized by granularity of the cell suspension, detectable when the tubes are gently agitated. If agglutination occurs in both tubes it is interpreted as auto-agglutination and as such has no significance. If the control tube is negative and the other tube positive the donor is incompatible. The readings should be made while the tubes are cold as the agglutination being comparatively weak may disappear on warming. The reaction is more definite if the tests

are conducted in tubes rather than on slides. The reactions are, as a rule, most pronounced at ice-water temperature. (Anti-Rh agglutinins capable of producing agglutination at 37°C. have been described.)

Summary

Haemolytic transfusion reactions may occur when both the donor and recipient belong to the same blood group. Since incompatibility of this nature can be detected only by direct matching of the bloods concerned such a procedure must always be carried out before a transfusion is given. Similarly in all cases where an interval of more than 10 days elapses between transfusions a fresh sample of recipient's blood must be matched with the prospective donor. It also follows that the same donor should not be used

a second time without again cross matching.

Most cases of intra-group incompatibility are due to the presence in the recipient's blood serum of an agglutinin designated "Anti-Rh". This agglutinin is best detected by tests conducted at ice-water temperature and these should form part of the routine matching procedure.

BIBLIOGRAPHY

- (1) Wiener, A. S.: Blood groups and blood transfusion. Springfield, Illinois. Charles C. Thomas. 2nd Ed. 1939.
- (2) Wiener, A. S., and Peters, H. R., Ann. Int. M., 1940, **13**, 2306.
- (3) Wiener, A. S., Arch. Path., 1941, **32**, 229.
- (4) Landsteiner, K., and Wiener, A. S., J. Exper. M., 1941, **74**, 309.

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HOSPITAL WOUND INFECTION IN WAR CASUALTIES

By R. J. WILSON, '42

Introduction

Many of the great contributions to medicine and its allied sciences have arisen from observations and studies carried out in time of war. The Great War of 1914-18, in which many of our senior colleagues in medicine and in surgery participated, gave to us several powerful weapons that are daily used in the offensive against disease and death, in peace and in war. The prompt use of Thomas's splint, the use of debridement, the extensive use of transfusions of blood and other fluids in the treatment of shock and haemorrhage are comparatively modern developments in traumatic surgery, yet these have already gone through their trial by fire and have proven themselves indispensable instruments in the hands of the surgeon whose lot it is to treat war casualties.

One of the problems that confronted the surgeon in the last war was that of severe pyogenic wound infection. Since that time a further weapon has been evolved with which he may combat this scourge, namely the sulphonamide drugs. However, before the end of the last war, and before the advent of the sulphonamide drugs, the foundation was laid for the study of this problem of wound sepsis, and efforts were made to control it by preventive measures. And although treatment of wound infections may be more satisfactory with the aid of sulphonamides the conceptions given to us by the bacteriological investigations on wound infection must not be disregarded, for what doctor would rather treat established disease than prevent its occurrence?

The Incidence of Haemolytic Streptococci in War Wounds

Fleming and Porteous studied the bacterial flora of infected wounds in hospitals in France and, in 1919, published their findings. They observed that in casualties arriving at a C.C.S.,

about 15 per cent of the wounds were infected with haemolytic streptococci. On arrival at the base hospital, when casualties were sent on immediately after operation, 20 per cent of the wounds were infected with haemolytic streptococci. However, after a stay at the base hospital of a week, over 90 per cent of cases had haemolytic streptococci in their wounds. The authors concluded that the increased incidence of haemolytic streptococci in wounds observed after a week in hospital was due to a hospital infection, the micro-organisms having been transferred from one patient to another probably during the dressing of the wound.

Stokes and Tytler (1918-19), the latter of whom was a Toronto graduate, published a paper in which they reported that 14.8 per cent of wounds in base hospitals in France contained haemolytic streptococci immediately before operation. Four to 14 days after operation the incidence of haemolytic streptococci in wounds had risen to 34.6 per cent. Thus the impression that many wounds were infected with pyogens, not at the time of infliction but sometime during stay in C.C.S. or base hospital, had been substantiated at that time by experimental evidence.

With the onset of hostilities in 1939, the problem was again brought into prominence in England by the large number of wounds requiring hospital treatment not only as a result of military activities but as a result of the large number of air-raid casualties among the civilian population. In light of certain work that had been done before the war, bacteriologists had methods available whereby they could study more adequately the problem of cross-infection of wounds in hospital. Miles and his colleagues, in 1940, published data obtained from studies in British hospitals. The incidence of haemolytic streptococcal in-

fection of wounds in casualties admitted to advanced base hospitals in a London sector averaged 25.3 per cent. However, in those air-raid casualties admitted without delay, the incidence was much the same as that given for 1918-19 by Stokes and Tytler, and by Fleming and Porteous, namely 14.3 per cent. In those Dunkirk casualties in which there was delay getting to hospital the incidence was 31.4 per cent. This latter figure tended to raise the average somewhat. They found it not unusual to obtain haemolytic streptococci from 70 per cent of wounds after the casualties had been in hospital for some time. The situation would appear to be a repetition of the state of affairs in 1918-19.

Another method of approach to this problem was undertaken by Miles et al. With the advent of Griffith's classification of the haemolytic streptococci in 1935, it became possible to study wound flora at various periods from the time of infliction until the time of discharge from hospital and to detect additions or change in wound flora. These investigations revealed the fact that some 58 per cent of wounds acquired new flora during treatment in hospital. The new species appeared on the average about 10 days after the first swabbing in hospital and 15 days after infliction of the wound. There has been no evidence comparable to that advanced in the case of tetanus that these micro-organisms can lie dormant in the tissues for long periods. The pyogens, on the other hand, and the haemolytic streptococcus in particular, usually become established rapidly and can be detected by bacteriological examination. There would seem to be little doubt that these micro-organisms are of hospital origin and in most cases were not introduced into the wound at the time of infliction.

Hare and Willits (1942) have recently examined the flora of 355 wounds within 2 hours of infliction. In none of these did they find haemolytic streptococci but in 4 cases the organism was isolated during convalescence. Hare has advanced the theory that nearly all strep-

tococcal infection of wounds results from the addition of the bacteria to the wound after its infliction. If this be the case, pyogenic wound infection should be almost entirely preventable. In order to prevent bacteria gaining access to the wound after its infliction it will first be necessary to know the sources of the micro-organisms, their manner of entrance into the wound, the channels by which infection may be spread in hospital wards and the effectiveness of measures designed to prevent the transfer of organisms from carrier to patient or from patient to patient. Considerable experimental work has been carried out on each of these phases of the problem, and they merit individual study.

Sources of Haemolytic Streptococci

Skin, clothing, etc.

Colebrook et al. in 1935 demonstrated that haemolytic streptococci, group A, are not normally found on the skin. Hare (1941) substantiated these findings, and in addition failed to isolate the micro-organisms from road dust, garden soil and clothing (except in the case of some nasopharyngeal carriers). It is unlikely then that any objects that might find their way into the wound at the time of its infliction would take with them haemolytic streptococci.

Nasopharyngeal carriers

Various figures have been given for the incidence of nasopharyngeal carriers in the general population—6.1% (Wright, 1940), 7.0% (Hare, 1940), 13.0% (Straker et al., 1939), 16.7%, 37.0% and 28.5% (Spooner, 1941). The wide variation may be explained by the fact that there is a marked seasonal incidence with its peak in the winter months (Paine, 1935). The figures are extremely high. It is little wonder that wounds tended by first-aid groups, or whoever else may be near, frequently infected on admission to hospital. These persons can obviously not wear masks, and when there is a chance of from 1 in 20 to 1 in 3 of the attendant being a nasopharyngeal carrier and infecting the

wound while examining and dressing it one would not be surprised at even a higher incidence of pre-operative infection. Add to this the fact that these nasopharyngeal carriers frequently infect their own clothes and hands (Hare, 1941) and it can be seen that droplet infection from the carrier is not the only method by which the dresser may infect the wound. Add to this the fact that the casualty himself may be a nasopharyngeal carrier and infect his own wound (a fact that has been borne out on investigation), and the figure of $15\% \pm$ for pre-operative infection does not seem at all improbable.

The risk of contact with nasopharyngeal carriers is not eliminated, however, on admission to hospital, for here it may even be intensified. There are many more to tend the wound, and unless precautions are taken the damage may be done for the first time, or further sepsis added. Firstly, the surgeon and his staff in the operating theatre may not have properly constructed nor properly adjusted masks. It has been shown by experiment that the type of gauze mask commonly used is not efficient in preventing droplet infection. In addition to this fact, many surgeons do not adjust their masks properly on the face, and labour under the delusion that provided a few layers of gauze cover the mouth, they may talk and perform an operation over a sterile field with impunity. This has been proven not to be so.

The greatest danger, however, lies not in the operating theatre, but in the wards. It is a sight worthy of citation to see a surgeon, junior or senior, or a dressing nurse, or a student, wear a mask while dressing a wound. The author has seen it occur once only. Why there should be any less rigid technique employed in exposing a wound in the ward for examination, for dressing or for clinical discussion, than is used in the operating theatre is beyond the comprehension of the average student of medicine. Yet it is done daily, and indiscriminately, regardless of the condition of the attendant's upper respiratory

tract. Therein possibly lies one important reason why the closed plaster treatment of compound fractures achieved such success within its own sphere of usefulness. It protected the patient from his attendants, and from frequent exposures to additional sepsis each time the dressing was changed.

Ward dust and air-borne infection

Cruickshank (1935), White (1936), Allison and Brown (1937), Willits and Hare (1941) have each given adequate experimental evidence that the air and dust of rooms in which infected wounds, puerperal sepsis, infected burns and scarlet fever cases are nursed contain large numbers of haemolytic streptococci. Thomas (1941) estimated that the dust from the morning's sweepings of a ward 4 days after a mild epidemic of streptococcal sore throat and follicular tonsillitis contained 102 million haemolytic streptococci. Spooner (1941) estimated that in the ward in which his investigation was carried out, haemolytic streptococci sedimented from the air at the rate of 6 per square foot per hour. These findings are representative of those made by many other workers of whom space does not permit mention. Every case of established infection can contaminate his bedclothes, the air and dust about his bed, and Willits and Hare (1941) have demonstrated that the contamination of the air is greatly increased by manipulation of the dressings, from which dried secretions are thrown out and carried for long distances by air-currents.

Hand and instruments

It is possibly not generally appreciated that a nasopharyngeal carrier or a patient with an infected wound, contaminates his bedclothes and person widely. It has been demonstrated that outer dressings which are frequently removed with the hands are heavily contaminated. In a busy ward, where many dressings have to be done and there is little staff to do them, streptococci might very easily be picked up inadvertently and carried to the next patient, and unless an

extremely rigid aseptic dressing technique is instituted, and adhered to in times of emergency, wound infection might assume almost epidemic proportions. This has been shown to be the case in certain hospitals in England where investigations were carried out.

Channels of Hospital Wound Infection

The channels through which pyogenic micro-organisms are spread throughout the wards have been investigated by studies on the haemolytic streptococci, because of the ease with which the individual types may be identified and traced from case to case. Okell and Elliott, in 1936, presented their findings on investigation of 14 outbreaks of streptococcal infection in otorhinological wards. They traced outbreaks from cases of mastoiditis and streptococcal sore throat. Spread of the organisms of the same type as the cases from which they originated resulted in cases of otitis media, prolonged pyrexia, scarlet fever and streptococcal sore throat. In some instances, doctors and nurses became infected. There was one death as a direct result of acquired infection in a healthy person. Many infections were latent and were revealed only by bacteriological examination. Keevil and Camps in 1937 presented similar findings from the investigation of 6 local outbreaks in a general hospital. There were examples of infection passed from nurse to patient, patient to patient, nurse to nurse. In one outbreak, an ambulatory patient was found to be a carrier and to spread infection throughout the ward. Allison and Brown, in 1937, demonstrated that 57 out of 100 patients in a scarlet fever ward harboured streptococci of a different serological type on discharge from hospital than that which they harboured on admission. Of 43 patients nursed in a multiple bed ward, 70.2% were reinfected. Fifteen per cent of the 33 showed no symptoms, but the micro-organisms were revealed by bacteriological examination.

From these investigations it is readily apparent that cross-infection in hospital wards is much more frequent than is

generally supposed. It is also apparent that a great deal of this is undoubtedly due to direct contact with a nasopharyngeal carrier, and that many of these carriers exhibit no signs or symptoms whatsoever. Ward attendants, whether they be doctors, nurses or orderlies, are liable to cross-infection from patients and in turn to reinfect other patients. Epidemics similar to those outlined above have been investigated in surgical wards in England. Miles et al., in 1940, and more recently Spooner (1941) have studied such outbreaks, with findings almost identical to those of the authors previously mentioned. They have also shown that although 70 per cent of wounds in a surgical ward may be shown to harbour haemolytic streptococci on bacteriological examination, only 5 per cent show clinical signs and symptoms of infection. However, each case in which the micro-organisms became established is another carrier to increase the hazard to new cases coming into the ward.

Other channels than that of direct contact with a nasopharyngeal carrier have been investigated. Mention has already been made of transfer of micro-organisms from infected cases by ward dust and air-currents. Hare et al. have demonstrated this fact experimentally both in the laboratory and in the ward as outlined earlier in the paper.

The question of transfer of organisms by hands and instruments through faulty dressing technique has been described. Little mention need be made of this channel of infection here, except to recommend for reference the improved dressing technique evolved in England and published by the Medical Research Council War Wounds Committee under the title of "The Prevention of Hospital Infection of Wounds".

Methods of Prevention

There is little need to elaborate upon methods of blocking the channels of cross-infection if all the possible channels are recognized and if the mechanism of cross-infection is clearly understood. Some of the principle points that have been em-

phasized by English workers may be briefly outlined.

1. Contact with nasopharyngeal carriers must be prevented by attendants wearing masks at all times when wounds are exposed for inspection or for dressing. The masks must be of such construction that they will prevent droplet infection. Staff attendants with an upper respiratory tract infection are not to report to duty until they have completely recovered from the infection.

2. Transfer of infection by ward dust and air currents may be minimized in many ways. Infected cases should not be nursed in the same wards as are clean cases. All sweeping, bed-making and dusting must be completed at least one hour before dressings are to be done. Spindle oil may be used on the floors to prevent dust from rising.

3. Ward-baths and other articles of common use are difficult to sterilize and should be watched carefully.

4. An improved dressing technique has been evolved, which utilizes a dressing team. All members wear masks. The details of dressing are outlined in the pamphlet, "The Prevention of Hospital Infection of Wounds".

If the recommendations made above seem to be impractical and unnecessarily complicated let it be borne in mind that in one English hospital where it has been in use, there has been a decrease in the number of wounds infected with haemolytic streptococci from 31.3% to 2.2% in head wounds, and from 4.4% to 0.0% in clean operative wounds over a 4-month period (McKissock et al., 1941). And

furthermore, until the importance of hospital wound infection is realized, especially by those of us who may shortly be working under conditions somewhat different from those obtaining in our teaching hospitals, any methods of prevention, even to the proper adjustment of a mask will seem unnecessary and impractical.

REFERENCES

- Allison, V. D., and Brown, W. A. (1937). *Jour. Hyg., Camb.*, **37**, 153.
 Colebrook, L., Maxted, W. R., and Johns, A. M. (1935). *Jour. Path. Bact.*, **41**, 521.
 Cruickshank, R. (1935). *Jour. Path. Bact.*, **41**, 367.
 Fleming, A., and Porteous, A. B. (1919). *Lancet*, **2**, 49.
 Griffith, F. (1935). *Jour. Hyg., Camb.*, **34**, 542.
 Hare, Ronald (1940). *Lancet*, **1**, 109.
 Hare, Ronald (1941). *Lancet*, **1**, 85.
 Hare, Ronald, and Willits, R. E. (1942). *Can. Med. Ass. Jour.*, **46**, 23.
 Keevil, N. L., and Camps, F. E. (1937). *Lancet*, **2**, 207.
 McKissock, W., Wright, J., and Miles, A. A. (1941). *Brit. Med. Jour.*, **2**, 375.
 Miles, A. A. et al. (1940). *Brit. Med. Jour.*, **2**, 855 and 895.
 Okell, C. C., and Elliott, S. D. (1936). *Lancet*, **2**, 836.
 Paine, C. G. (1935). *Brit. Med. Jour.*, **1**, 243.
 Spooner, E. T. C. (1941). *Jour. Hyg., Camb.*, **41**, 320.
 Stokes, A., and Tytler, W. H. (1918-19). *Brit. Jour. Surg.*, **6**, 111.
 Straker, E., Hill, A. B., and Lovell, R. (1938). Ministry of Health Pub. Health and Med. Subjects Rep., No. 90, London.
 The Prevention of "Hospital Infection" of Wounds (1941). M.R.C. War Memorandum No. 6, London.
 Thomas, J. C. (1941). *Lancet*, **1**, 433.
 White, E. (1936). *Lancet*, **1**, 941.
 Willits, R. W., and Hare, Ronald (1941). *Can. Med. Ass. Jour.*, **45**, 479.
 Wright, J. (1940). *Jour. Hyg., Camb.*, **40**, 647.

DEFORMITIES OF THE SPINE

By A. T. JOUSSE, B.A.

Apart from traumatic lesions, deformities of the spine are not surgical emergencies and are therefore subject to treatment following a careful consideration of the individual case when the optimal time for surgical intervention may be chosen with care. Treatment is not so much a life-saving measure as it is directed toward the re-establishment of the patient as a useful member of society who may live his allotted span fairly free from pain and discomfort.

The following lesions which do not completely cover the field will be considered.

CONGENITAL DEFORMITIES

1. *Spina Bifida*.

This is the incomplete closure of the vertebral arches with or without an accompanying protrusion of the meninges or part of the cord. When the contents of the canal are not displaced, the skin surface is intact and there are no neurological findings, though the dorsal arches are incompletely united the condition is called *Spina Bifida Occulta*. This condition is usually recognized as an incidental finding in radiography or at autopsy.

No treatment is indicated.

Sometimes *Spina Bifida Occulta* is not occult but is revealed by the presence of surface markings as a cicatrix, a patch of pigment or a growth of hair at the skin surface over the deeper lesion. *Myelocoele* is the condition when there is exposure of the spread out cord structures at the upper end of which the central canal opens. It is usually in the lumbo-sacral region.

It is incompatible with life beyond a day or two.

In *Meningocoele* the meninges are exposed at the surface. The cord is in its normal position. Neurological signs and symptoms may or may not be present.

Meningo-myelocoele and *syringo-myelocoele* are variants of the above. In the first the cord structures follow

the bulging posterior wall of the meningeal cavity; in the second the central canal is dilated so that the posterior wall of the cord is in contact with the bulging membranes, the anterior wall is normally situated. The lesion must be above the level of lumbar two.

Etiology

The cause of congenital lesions is unknown but the mechanism whereby deformities are produced is understood, in this case at least.

The nervous tissue in the embryo arises from the epiblast. As growth proceeds the mesoblastic layers intrude between the nerve tissue and the skin on the dorsal surface. From the mesoblastic tissue the vertebral muscles, ligaments and bony structures form. Two centres of ossification for each neural arch are developed from this tissue, one centre being situated in each half of the arch. These centres should successfully build the arches (laminae and pedicles). Sometimes they fail to do so with a resulting *Spina Bifida*.

Signs and symptoms vary from very evident physical and neurological abnormalities to total lack of clinical evidence of deformity. There may be the Hall Mark on the skin surface as described above. The meninges and associated structures may bulge out; the protrusion may be pedunculated or on a broad base. Ulceration of the protruding tissues is common. The site varies from the sacrum to the skull but is most common in the lumbo-sacral region which is the last part of the vertebral arch to fuse.

A radiograph will invariably reveal the defect in the bone. Neurological findings of the nature of a paraplegia with spasticity are suggestive of this lesion. Occasionally a *Spina Bifida Occulta* is unsuspected until in adult life low back pain develops.

Treatment

If no signs or symptoms develop no

treatment is necessary or desirable.

Protruding structures should be protected from pressure and irritation by pads or bandages.

If ulceration threatens the protruding part must be cut away, the neck of the sac being tied.

Should nervous tissue be included in the sac this must be dissected out and replaced in the canal, often with skin attached. The erector spinae muscles are drawn together in an effort to correct the defect.

Prognosis

Hydrocephalus and meningitis are frequent sequelae to operation. William Cone of the Montreal Neurological Institute points out that removing the sac removes absorptive tissue. If the substance of the sac can be preserved and replaced in the canal hydrocephalus can be obviated in some but not all cases.

At the Mayo Clinic operative mortality is 50% (Craig).

Cone gives an operative mortality of 14% by operating at 3 weeks whereas at the Hospital for Sick Children, Toronto, operation is deferred until the third year.

In any case neurological lesions, once established are not remedied by operation.

2. *Wedged Vertebrae.*

The vertebral bodies of one or more segments develop unequally on the two sides thus forming a wedge-shaped vertebral body. Should it occur in the thoracic region there will be no rib on the narrow edge.

The result is scoliosis, easily recognized clinically and the cause of the curvature is revealed by the X-ray. The scoliosis progresses until growth is completed when the curves become fixed.

Treatment as for Scoliosis

3. *Sacralization of the Fifth Lumbar Vertebra; Fused Vertebrae, and Hemi-Vertebrae are deformities encountered.*

The diagnosis is often made when investigating the cause of an obscure low back pain.

Any course of treatment deals with

symptoms as such for the cause is irremediable.

4. *Spondylolisthesis.*

This is a forward, displacement of one vertebral body on the next lower one. It occurs most commonly at the lumbo-sacral junction.

Etiology

Fracture of the articular processes or congenital weakness of the pedicles of the vertebrae which allows the laminae and articular facets to remain in their normal position while the detached vertebral body slips forward.

Signs and Symptoms

If displacement is slight there will be only local pain; if to a marked degree there will be neurological signs and symptoms caused by pressure on the cauda equina.

Diagnosis: The patient is brought to his medical attendant by pain and weakness in the back and legs. The lumbar lordosis may be accentuated the stature thereby shortened. X-ray findings are conclusive.

Treatment

Prolonged rest in the recumbent position; later, crutches may be used to transfer the weight of the body to the axilla thus relieving the lumbar spine of stress. Albee's operation for spinal fusion is sometimes advisable. In this operation a bone graft from the tibia is wedged into slits made in the tips of the spines of the vertebrae to be fused. Wire sutures fix the graft in place. Immobilization in a body cast for two or three months follows this operation.

Watson-Jones advocates manipulative reduction of the displaced vertebral body. The patient is placed on his back. He is supported only by his shoulders which rest on a table and by the legs, hip and knee joints being flexed to ninety degrees.

Gravity alone, acting on the body or augmented by a weight applied to the hypogastrium reduces the lordosis. A body cast is applied in this position (including the legs) and left in place for five months.

Prognosis

Watson-Jones believes reduction is impossible in a patient over fifteen years of age and equally impossible in any patient in whom the condition has existed for more than two years irrespective of age.

ACQUIRED LESIONS

5. *Traumatic.*

Fractures of the vertebral column may be complete or incomplete.

A complete fracture, also known as a fracture dislocation, as the term implies, involves fracture of the vertebral structure with dislocation of one body from the next. Almost invariably there is cord damage associated therewith.

An incomplete fracture is one where any part of the vertebra is fractured but where there is no dislocation at the articular facets. Cord damage may or may not be present.

Etiology or Mechanism of Fracture Production

The violence may be direct or indirect, the latter being the more common. Direct injuries are caused by violence directly over the vertebral column or by gunshot wounds. Such trauma frequently damages the spines of the vertebrae and the structures about the neural canal excluding the vertebral bodies. A sudden muscular contraction as of the psoas or the quadratus lumborum can fracture a lumbar transverse process.

Indirect violence acts when a person falls from a height, landing on his feet or when a heavy weight falls on his shoulders. The resulting forcible flexion of the spine causes a crushing injury to one or more vertebral bodies. Those vertebral bodies situated at the junction of a relatively immobile and a mobile segment of the column as the dorsi-lumbar region or the cervico-dorsal region are more susceptible to injury.

Depending on the degree of violence there may or may not be dislocation of one vertebra from another. The verte-

bral body may be compressed toward the front or to one side. The articular processes may be broken or dislocation may take place without fracture of these structures. There may be rotation of the vertebrae.

Gunshot wounds may, of course, produce a variety of lesions in the bony structure with or without damage to the cord. A missile passing in proximity to the vertebrae but not damaging them may cause cord damage.

Signs and Symptoms

There may be a localized area of swelling and tenderness; there may be deformity with a bony prominence. Great pain on attempted movement is a common finding. If cord compression or injury is had there will be neurological signs. Occasionally a lumbar body is compressed with no resulting disability or at most referred pain suggesting an intra abdominal lesion.

The diagnosis depends on the X-ray findings both antero-posterior and lateral plates being necessary.

Treatment

Injury to transverse and spinous processes can be well treated by confining the patient to bed for three or four weeks in the first case and by strapping in the second.

For a compression fracture of the dorsi-lumbar region if the signs of cord injury are absent or minimal the anaesthetized patient is fixed in hyper-extension in a body cast for twelve weeks. The cast must extend from the great trochanters to the axillae. Spinous processes and other bony prominences should be protected by pads. After the cast is removed the patient may be up in three weeks but a spinal support is often necessary for some time.

For fractures of the cervical region a cast is also applied with the cervical vertebrae in the position of extension but the head should not be extended on the atlas. The cast must encase the chest and neck and must prevent head movement. To this end a band of plaster surrounds the forehead.

Operative Treatment

In general surgery is not indicated for if there is cord injury function will not be restored; if there is haemorrhage the exudate will be absorbed through time and oedema will be resolved. Some advocate operative intervention when the cauda equina is injured for it has regenerative power or when a spicule of bone penetrates the cord. Again it may prove effectual when severe intractable root pain is present.

Decompression by laminectomy is done in some instances.

Operative procedure of the type required is very difficult and does not offer a better prognosis than conservative treatment.

Fracture Dislocations

Fracture dislocations are more serious and respond less readily to treatment. If severe they are readily recognized by angular deformity, paraplegia and X-ray. Laminae are often injured and intervertebral ligaments torn. A cervical region dislocation is commonly unilateral, the lower border of the inferior articular process becoming caught in the superior intervertebral notch of the vertebra below. The head assumes a characteristic attitude with chin lowered and turned to the opposite side. There is restricted movement with pain caused by pressure on nerve roots. One or more of the articular facets may be fractured.

Treatment

Reduction may be effected by manipulation or open reduction. For the former the articular processes must be intact. The neck must not be hyperextended. In a small percentage of cases operative intervention is successful. Its object is fixation of the dislocated parts after reduction. Some type of operation like the one devised by Albee is used.

Manipulative reduction under anaesthesia may be tried if the lesion is not too high, so high that any further cord damage will lead to a fatal outcome. In some cases reduction occurs as soon

as the patient is placed flat on his back but re-displacement occurs just as readily on movement.

A fracture of the atlas may occur when a fall on the head is suffered. The anterior and posterior arches suffer damage.

There is spasm of the neck muscles with pain.

Treatment

Skeletal head traction with the application of a plaster fixing the head in the mid position for three months is one method of dealing with this condition.

A fracture of the coccyx is caused by a fall in the sitting position. Pain on sitting and on defaecation are indications of the injury. The history and X-ray confirm the diagnosis.

Treatment

Union may require three or four months. Removal of the coccyx is the only successful treatment in some cases.

Prognosis

The ultimate result depends largely on the presence or absence of cord damage. In the dorsal region the canal is narrowest and a slight displacement may cause paralysis whereas in the lumbar region a marked displacement may produce no neurological lesion. The danger decreases from above downwards. If there is no cord injury there should be no mortality. Residual symptoms of fatigue and pain may persist for the remainder of the patient's life. Sometimes bone grafts are required though bony union is the rule. With cord damage if the patient survives two weeks his chances are good for prolonged survival.

Apart from the immediate injury death results from pneumonia, pyelonephritis secondary to a cystitis or from infected bed sores. Watson-Jones says that functional results are perfect in two-thirds of his cases there being no permanent stiffness or pain and the back is as strong as before the injury. This excludes cervical lesions.

The average period of incapacity is seven months for those engaged in sedentary occupations and ten months if engaged in heavy work.

W.C.B. cases frequently require two years before returning to work as the personal element enters into the recovery period when no pecuniary loss is being suffered.

Dislocations as such occur only in the cervical region owing to the position of the articular processes and the relative lack of fixation of the cervical column. The dislocation may occur between any two cervical vertebrae including the occiput from the atlas. However the most common site is between five and six. The cause is forcible flexion of head and neck, combined with rotation. The lesion may be uni-lateral. If bi-lateral the cord damage is usually severe with fatal outcome.

Treatment

There is little that can be done to avert a fatal outcome if the lesion is bilateral. Traction on the head with the neck flexed may be tried and if not successful in effecting reduction an open reduction is the only alternative. It may be necessary to excise part of the articular facets of the lower vertebra involved in order to effect replacement of parts. A recurrence of the dislocation is very likely to follow unless some type of spinal fusion operation is also done.

The reader must bear in mind that the treatment of fractures of the spine begins with prevention of further damage to the cord and precautions must be taken at the scene of the accident for if through inexpert handling a fractured spine without serious cord damage be converted into one with a severed spinal cord not all the Piety nor Wit of man can serve to erase the damage which may, for all practical purposes, sever a man from half of his body—a very gloomy thought.

Admittedly it is no easy matter to take the precautions for the first aid treatment of spinal injuries, which are outlined in all first aid manuals, when at the scene of an accident. Calmness

is at a premium; there is a seemingly homogeneous mass of damaged vehicles and human bodies which must be disentangled and there is an excess of willing but unskilful lay assistance. Nevertheless a brief examination and questioning of the injured if conscious and the treating of unconscious and doubtful cases as cervical fractures may possibly prevent a few undamaged cords from being ruined. If care can save even one person from physical incapacity the precautions are more than justified.

It should be remembered that since the advent of the sulfonamide drugs the likelihood of a bed-ridden patient being released through the agency of an intercurrent infection has been much reduced.

6. *Retropulsed Intervertebral Discs* (*Extruded Nuclei Pulposi*)

This topic has been dealt with fully and efficiently in a recent article in this journal (March 1941) so will not be discussed here.

Suffice it to say that all are not in agreement as to the prominent part this lesion is said to play as a cause of disability.

7. *Scoliosis*.

By definition this is a lateral curvature of the spinal column often associated with rotation of the vertebral bodies one on another. There is a primary curve, the only real deformity and two secondary or compensatory curves which maintain the head erect.

Etiology

1. Congenital.
2. Paralytic.
3. Idiopathic or postural.
4. Miscellaneous—empyema, thoracoplasty, fracture, tuberculous, etc.

1. Congenital. Caused by the presence of wedge-shaped vertebrae (see above).

2. Paralytic. A unilateral weakness of the muscles which hold the trunk erect may result from anterior poliomyelitis infection. The condition progresses until the period of growth is completed. The

convexity of the primary curve is to the paralyzed side.

3. Idiopathic. This is the most common type. It is more common in girls than boys usually being found in the type who are of poor muscular development. The age of onset may be early or late but reaches its peak incidence between the ages of seven and nine years, and progresses until growth ceases.

Signs and Symptoms

If marked in degree it is obvious to the observer.

The rotation of the thoracic vertebrae causes the ribs to be very prominent on one side posteriorly, and flat on the other. This condition is increased when the patient bends forward.

One shoulder is higher than its mate and the trunk is shortened. The iliac crests are at the same level but one is more prominent than the other and the prominent one appears higher. The more prominent one is on the side opposite to the convexity of the primary curve.

Pain is rarely present but fatigue is marked and if the condition is so severe as to embarrass the action of the heart and lungs impairment of health may result.

Diagnosis is made by physical examination, history and X-ray.

4. The miscellaneous types are likely to be less severe and when recognized are treated best by attacking the underlying cause.

Treatment

In the early stages the idiopathic group can be improved by following prescribed exercises aimed at improving posture and not directly at straightening the spine. In more severe cases exercises may prevent advancement of the condition though may fail to correct the curvature. If the primary curve can be eradicated in any way the compensatory curves correct themselves. To achieve this the primary curve must first be determined. This is not always easy. Usually the primary curve is the largest and often it is the middle one.

Invariably it is the most rigid. To identify it three flat plates must be taken. The first is with the patient in the erect position; the second with the trunk bent as far as possible to the right side and the third with the trunk similarly bent to the left. The curve which changes least in the three positions is the primary curve.

The next step is to straighten this primary curve as much as possible. This is achieved by suspending the patient in a hammock-like bit of apparatus, by one arm and both legs, for progressively longer periods each day. The side which is uppermost is the one toward which the convexity of the primary curve is directed. When this curve has been reduced as much as it is thought possible to reduce it the patient is encased in a body cast of plaster of paris. The head and one leg are included. The position of the patient is that which he was in when suspended in the hammock. After two weeks in the cast during which time one determines whether or not the patient's morale is adequate to sustain him during his stay in this extreme position a hole is cut in the back of the cast and a spinal fusion operation is done using a bone graft from the tibia. The graft must extend from vertebrae above to vertebrae below the lesion whose axes are vertical. The cast is left in place for three and a half months after the operation. A brace or supporting jacket is worn for from six to eighteen months after the removal of the cast.

Prognosis

Perfect results are never obtained but improvement is often worth the effort particularly in paralytic cases.

The health and morale of the child must be good to withstand such prolonged confinement in a cast. It is doubtful if adults could stand such treatment.

DEFORMITIES CAUSED BY INFECTION

8. *Tuberculosis of the Spine* (Pott's Disease).

The tubercle bacilli are lodged in the

vertebral bodies in this disease and cause the usual destructive lesion. Children are frequently the victims. The centre of the vertebral body becomes caseous; the disease involves and destroys the intervertebral discs (a point of diagnostic value to the radiologist) but spares the transverse processes, the spines and articular processes. As a result the bodies collapse in front, the arch surrounding the cord does not and a degree of kyphosis results.

Signs and Symptoms

There is general evidence of tuberculosis. The kyphosis may be very evident. Neurological signs may result from pressure on the spinal cord resulting from the deformity or from the excess of granulation tissue or so-called tuberculous pus pressing backward the posterior common ligament of the spinal column.

Diagnosis depends on the history, the physical examination and the X-ray. The discs may be destroyed in this condition but are intact with tumor growth (metastatic) in the vertebral bodies.

Treatment

There is no specific for infection with the tubercle bacillus.

The best results are obtained when perfect rest is had, thus enabling the healing processes of nature to act most effectively.

The following course of treatment is the one in use at the Weston Sanitarium. The patient is put to bed for six months to rest the lesion so that it may become quiescent as surgical intervention is contraindicated on an active focus. During this time the kyphotic deformity is corrected by keeping the patient on his back on a Whitman frame in which there is a gradually increased angulation so that hyperextension is finally achieved. When at the end of six months clinical and laboratory findings indicate that the infection is quiescent a spinal fusion operation is done. This is followed by four to six months more confinement to bed. Next comes a convalescent period of three months.

Thirty per cent of the patients so

treated are hospitalized for fifteen months. Another thirty per cent for two years and the remainder for an indefinitely longer period.

Prognosis

This method gives the best results yet obtained and many patients are restored to a useful place in society.

MARIE-STRÜMPPELL SYNDROME (SPONDYLITIS DEFORMANS)

This is a progressive disease wherein the articular facets and the vertebral bodies become united by bony ankylosis. In addition ossification of the anterior longitudinal ligament takes place. The spine becomes a rigid column. As the disease progresses the normal curvatures of the spine disappear and the whole arches forward until the patient is flexed and immobile in a bowed position with chin on chest. There is severe pain associated with this disease. It attacks the young as well as the old and progresses over many years.

Etiology

Some unknown infective agent to toxin is thought to be the cause. It is interesting to note that a similar disease attacks the workers in cryolite factories in Denmark where fluoride compounds are used as solvents for bauxite ore. It is thought that in this case fluoride poisoning is the cause.

Diagnosis is based on a history of progressive curvature of the spine accompanied by pain and disability. The X-ray reveals the presence of the bony ankylosis and the ossified ligaments.

Treatment

The curvature of the spine is reduced in the same manner as the kyphosis is reduced in Pott's Disease. This necessitates a prolonged stay in bed on an angulated Whitman frame. The duration depends on the ease with which the curvature can be reduced. It is at least three to six months. A spinal fusion operation is done in two stages the end result being a straight rigid spine; the fusion involves the cervical, thoracic and lumbar segments.

Prognosis

Treated or untreated the patient has a fused spine, ultimately. However, in the treated cases the spine is straight. In the untreated it is curved and the victim is incapacitated; he suffers much pain and discomfort over a period of many years whereas surgical intervention reduces the period of discomfort to one or at most two years and turns out an individual who is able to be self-supporting in society.

Many other deformities of the spine exist but space forbids discussion of the rarer conditions.

BIBLIOGRAPHY

- (1) R. Watson-Jones: Fractures and Other Bone and Joint Injuries, Ed. II.
 - (2) George Wilson: Fractures and Their Complications.
 - (3) Rose and Carless: Text Book of Surgery.
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PHOTODYNAMIC ACTION

By C. W. J. ARMSTRONG, '44

The presence of small amounts of certain foreign substances, called photosensitizers, in living tissue renders that tissue sensitive to visible light. Photodynamic action has been defined as the chemical changes produced in such a sensitized tissue upon exposure to visible light; by definition molecular oxygen must take part in the reaction (Blum, '41). That photodynamic action can affect a wide range of biological material will be evident from a list given below; these changes include inactivation of enzymes, hemolysis, killing of tissue, etc.

Photodynamic action has been shown to be the fundamental process in a number of interesting diseases characterized by photosensitization. Indeed, this principle of photosensitization is used in the production of modern photographic film; in this case a photosensitizer eosin, say, is added to the silver emulsion to render it sensitive to wavelengths to which it is otherwise relatively insensitive.

History

About 1898 Oscar Raab was studying the toxicity of the dye acridine on paramecia; he observed that the acridine was relatively more toxic in the light than in the dark. Light alone (no dye) had no effect on the organisms. Thus the combination of light and dye was necessary for the effect. This phenomenon was termed photodynamic action. Raab also observed that the toxicity of the dye was related to the intensity of the light, and that light previously passed through a filter of the photosensitizing substance had no lethal effect on photosensitized paramecia. (Thus the initial step in the process is the absorption of light by the photosensitizer.) These findings stimulated considerable interest and many papers on this subject appeared during the following decade.

In 1905 Sacharoff and Sachs reported that reducing agents, e.g. sodium sulphite, inhibited photodynamic hemolysis. Metz-

ner (1919) demonstrated that the killing of protozoa by photodynamic agents required the presence of oxygen. Efimoff (1923) showed that the initial photodynamic reaction is irreversible when he reported that the killing of paramecia by eosin and light required the same amount of light regardless of whether the irradiation were continuous or intermittent.

During the last 40 years many types of biological system have been reported to be susceptible to photodynamic action. A representative list of such effects is given:

- denaturation of proteins
- permeability changes; hemolysis; cytolysis
- inhibition of blood clotting and of leucocyte movement
- stimulation of: muscle (smooth and voluntary); plant growth
- inactivation of: toxic organic compounds, hemolytic snake venoms
- bacterial toxins and various kinds of antibodies
- viruses, bacteriophage
- enzymes
- destruction of vitamins and hormones
- death in: bacteria and higher plants
protozoa; coelenterates, nematodes, annelids and echinoderms; vertebrates.

Chemistry

The substances which are known to be capable of acting as photodynamic sensitizers fall into several groups when classified according to their chemical structure. All these agents are carbon ring compounds and are coloured (so that light is absorbed by them). Some of them are:

- (1) Dyes of the triphenylmethane type —fluorescein and its halogen derivatives eosin, erythrosin and rose bengal; also mercurochrome, phloxin.

(2) Members of the naphthalene and anthracene series—quinine, cyanine; anthraquinone, acridine, flavines, methylene blue.

(3) Porphyrins and related compounds—blood pigments, chlorophyll and their derivatives; bile pigments.

(4) Compounds of unknown constitution found in plant material.

To produce photodynamic action wavelengths which are within the region of the absorption spectrum of the photosensitizer employed, and of sufficient intensity, must be used. Molecular oxygen is also required by the photodynamic reaction; the latter can be inhibited by the presence of reducing agents or by the absence of oxygen. Blum has shown that iodide ion can be photodynamically oxidized. Photodynamic action can be inhibited by the presence of certain aromatic compounds also; these may act by having a sparing action on the substrate in the tissue which is normally oxidized by the photodynamic agent. Just what this oxidizable substrate is, whose destruction results in the variety of effects attributed to photodynamic action (listed above), is not known. For various reasons it is believed to be protein in nature.

The photodynamic reaction is irreversible, has a low Q_{10} (about 1.1, which is characteristic of photochemical reactions), and has an extremely low R.Q. (0.047).

All known photodynamic agents are fluorescent but the significance of this property is not understood. No quantitative relationship between fluorescence and photodynamic efficiency has been shown. Many of the effects of photodynamic action can be simulated by (1) using the photosensitizer in the dark in much higher concentration, or (2) the use of ultraviolet light with no photosensitizer. These two types of reaction are independent of oxygen.

H. F. Blum has been very active in this field during the last ten years and he has done much to clarify our views on the mechanism of photodynamic action. In a worthy attempt to clear up the confused maze of evidence concerning photodynamic action, Blum has sought to de-

fine photodynamic action by setting up three provisional postulates (Blum, 1941, p. 131) which the reaction must satisfy in order to be classed as photodynamic action. They are:

(1) "The symptoms of light sensitivity must be elicited by exposure of the animals to sunlight, preferably to sunlight through window glass.

(2) A photodynamic substance must be isolated in pure form, which will produce the symptoms if injected into the experimental animals only when followed by exposure to light.

(3) It must be demonstrated that the wavelengths which produce sensitivity in postulates (1) and (2) are identical."

Blum has worked extensively with the photodynamic hemolysis of erythrocytes by rose bengal. Some of his principal findings are summarized here:

(1) The dye does not appear to be adsorbed in the ordinary sense by the tissue.

(2) The degree of effect depends upon the concentration of absorbed dye.

(3) All dyes require about the same amount of radiant energy per absorbed molecule of dye to produce a given degree of effect. Hence the reason one dye is more effective photodynamically than another appears to be because it is more completely absorbed.

(4) One molecule of absorbed dye can take part in the oxidation of many molecules of the susceptible part of the cell membrane.

Photodynamic Action in Disease

(1) *Etiological*—From a number of diseases or conditions attributed to photodynamic action a few, about which relatively more is known, will be selected for mention. In general, those photodynamic diseases which affect animals, resulting in obvious economic loss, have received more thorough study than those occurring principally in man. The source of the photosensitizer is, in nearly all cases, plant material. Hence photosensitization is not infrequent among farm animals (in some parts of the world) for they are not only

largely herbivorous, but they also sleep and spend most of their spare time in contact with plants. Most of these diseases are associated with a dermatitis and perhaps edematous swellings in the unpigmented areas of the skin of animals and in those areas of human skin normally exposed to light.

Internal—These are usually the result of ingesting a photodynamic agent (1) normally present in some plant material used as food, or (2) in certain drugs taken internally.

The condition called Hypericism is due to the presence in the skin of a fluorescent substance, or substances,—“hypericin”; this principle can be extracted from the common St. John’s-wort (*Hypericum perforatum*). This plant, originally European, is widely distributed in southern Ontario. It occasionally contaminates forage with serious results, especially in sunny weather. Highly pigmented animals are, of course, better protected.

A similar disease is Fagopyrism which is due to the ingestion of large amounts of buckwheat (*Fagopyrum esculentum*), and subsequent exposure to light. Recently Chick & Ellinger (1941) have shown that a fluorescent photodynamic principle can be extracted from the hulls and flowers of the buckwheat plant. This disease occurs in farm livestock but seems to be practically unknown in man. Possibly this is because man consumes buckwheat after its manufacture into flour, and from this product the hulls are largely removed during the process of milling (Leach, 1920). (It is perhaps of interest to note that buckwheat hulls have been used as an adulterant in black pepper, Winton, 1932.)

Geeldikkop (yellow thick head) is a disease which is of considerable concern to the sheep ranchers of South Africa and the southwestern U.S. In this disease a plant poison impairs liver function, so that a porphyrin, phylloerythrin—a breakdown product of chlorophyll, normally excreted in the bile—is retained in the body and photosensitizes the animal. The sheep are jaundiced and have swollen

lesions about the face, whence the name of the disease.

Drugs such as quinine, flavines, sulphanilamide, etc., sometimes seem to photosensitize the patients to whom they are administered.

External—These are contact agents which have been applied to the skin intentionally or by accident. Oil of bergamot which is widely used in cosmetics, e.g. lotions and creams, sometimes photosensitize the users. An active substance has been extracted from figs which may explain the occasional photosensitivity of fig handlers. Cases have also been reported of asphalt workers becoming photosensitized. Drugs which have been applied externally, e.g. flavines, impure vaseline, eosin (as a bromine reagent) have resulted in photosensitization of the patient.

Some investigators believe that chlorophyll acts as a photodynamic agent, but there is some evidence against this view. Some of the above reports are not believed to be cases of true photodynamic action, but represent other types of photosensitization; this is so for sulphanilamide and for chlorophyll.

(2) *Clinical*—In the literature there are reports dating from well back in the eighteenth century which describe conditions in animals closely resembling photosensitization. At that time, for lack of a better explanation, such conditions were usually ascribed to the mysterious powers of lightning, etc.

Light sensitivity is now known to be a factor of relative unimportance in the deficiency disease pellagra. The sensitivity in pellagrins may be due to the presence of a porphyrin. Much has been written regarding the photosensitizing action of blood-derived porphyrins, especially hematoporphyrin: their importance seems to be overrated as there is frequently no relation between the presence or absence of porphyria and light sensitivity in disease. The evidence on this point is very contradictory.

A number of conditions are known clinically in which a dermatitis is associated with a photosensitization, e.g.

Hydroa. There will not be space to discuss these. The etiology of some of them is poorly worked out, making their recognition difficult and leading to confusion in terminology and interpretation (Blum, 1941). A cursory examination of the histories of twelve cases of Dermatitis venenata treated in the Toronto General Hospital during 1941 revealed that sensitivity to light is mentioned in none of them. Three of the twelve histories, however, suggest that photosensitivity may have been present.

In conclusion, more investigations of an experimental type are clearly needed

in order to relate the clinical findings to the facts which are being brought to light by fundamental research in this field.

REFERENCES

- Blum, H. F., 1932: *Physiol. Rev.* **12**: 23.
 Blum, H. F., 1941: "Photodynamic Action and Diseases Caused by Light". A.C.S. Monograph No. 85.
 Chick, H., and P. Ellinger, 1941: *J. Physiol.* **100**: 212.
 Leach, A. E., 1920: "Food Inspection and Analysis".
 Mathews, F. P., 1937: *Arch. Pathol.* **23**: 399.
 Winton, 1932: "Structure and Composition of Foods".

NOTE: References mentioned in the text but not listed above can be obtained from either of the excellent reviews of Blum or from that of Matthews.

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Feature Section

J. B. ROBERTS, Editor

THE SHAPE OF THINGS TO COME: A LETTER FROM THE PRESIDENT OF THE ONTARIO MEDICAL ASSOCIATION

Toronto,
December, 1941.

Dear Mr. Roberts:

I have your letter of Dec. 8, 1943, asking me to write a feature article for the University of Toronto Medical Journal. Let me say at once that I am not an experienced "feature-writer". I shall make an attempt, however, to discuss in letter form some of the subjects engaging the attention of the Medical Profession, including undergraduates, today, in the hope that the attention of the undergraduate body may be focused on the subjects and problems which will so vitally affect their future.

One subject that is provoking a good deal of comment is Medical Education. As the process of education is not static, students and graduates do well to consider the path they are treading or have trod, and whither it is leading and likely to lead in the future.

It is conceded by the majority that undergraduate training leaves little to be desired from a scientific point of view. There is criticism of our medical education from other standpoints. To these criticisms attention should be paid. If they are not answered, or the conditions giving rise thereto not explained or corrected, some students will leave the University disgruntled, perhaps disloyal. Such sentiments detract from the esprit de corps which should vitalize any body of University graduates.

One criticism not infrequently heard is that "students never get to know their teachers". In a University with a large student enrolment that is only too true. This is unfortunate, as it is conceded that both parties lose much where intimate association and genuine interest is handicapped. This is particularly true of the medical profession, where intimate association with people is as essential as our daily bread.

Employers are beginning to recognize this close association as essential in industry. An employer recently said: "I have found that what is needed in most cases is a change in our attitude towards men. What it amounts to is, that besides not being indifferent, we have to have a real caring for them." Now it seems to me that what applies to a large industry applies to a large university. From the highest to the lowliest member of the teaching staff, there must come greater knowledge, sympathy, and understanding of individual students, if we as teachers are to get the best work from them. Anything that causes friction and is not corrected is likely to have a deleterious effect on the work of all. In industry this friction, not wages, is said to be the fundamental cause of strikes. In the university it is the cause of poor work and disloyalty.

Therefore any steps taken to correct this defect should be welcome. In the past it seems that any attempts to establish a more intimate relationship between students and teachers have been more or less sporadic. It would seem reasonable that more definite steps should be taken in the future, by a free discussion of this problem separately and conjointly by students and teachers alike.

Down through the ages students have been very critical of their teachers. This is natural. It is caused by several factors. There are natural antipathies best explained by the

"I do not like thee Dr. Fell,
The reasons why I cannot tell."

All teachers and lecturers are not endowed with the same inherent qualities for teaching. Few have nowadays had any training in the science of pedagogy. This used not to be so, as so many had taught school before coming to the University. This lack of training is a distinct loss to would-be teachers, and should be given consideration in the teaching of medicine in the future.

One professor (now retired) with a long experience in teaching, put it quite succinctly quite recently when he said, in substance, "There should be fewer teachers, and they should be well-trained, and adequately paid". This would give more time and leisure for the planning and giving of a course of instruction, free from the demands and irritations of private practice. More time would then be given to a sympathetic understanding of students, their background, environment, and academic standing. So much time would not be wasted, in the clinical years at least, in classifying the members of a class, academically and otherwise. There would be less tendency to "mass production", more time for quiet reflection for both parties concerned. Therefore the time may come when there will be more teacher-specialists, and fewer specialist-teachers—the latter a mistake where the primary objective of University training in Medicine is the production of general practitioners.

Post-graduate instruction has now come to be regarded as a necessity. It has become almost an essential for a new graduate in Medicine to spend one and not infrequently two or three years in post-graduate work. This is all to the good, but there is a tendency for an ever increasing number of graduates to be specialists, and to start practice as such. That is not desirable. The body does not exist as so many water-tight compartments. I for one would subscribe to the late Sir James MacKenzie's view, that none should be a specialist who has not spent some time in general practice. (He said ten years). This is a good fundamental training for everyone who intends to practice, no matter what his objective, as it leads to a better understanding of persons and the way they should be managed. Experience too is gained in conducting a business, a very necessary training in these days of economic stress and changing social conditions, with all the ramifications arising therefrom, demanding a strict accounting in a legitimate business. Therefore no matter what post-graduate work is held in perspective, make the foundation for such secure. Even during undergraduate days, if possible seek out the opportunity of seeing, in association with a good general practitioner, something of the practice of medicine as it is carried out in the home, and outside of large hospitals. Resourcefulness, ingenuity, as well as a knowledge of the science of medicine, come into play. By such an experience the specialist and the general practitioner alike will be spared many a heartache, and patients will be viewed as human beings, not just as "cases".

Throughout the professional careers of all, postgraduate study must go on. The science of medicine advances rapidly and facilities for further instruction must be available. For many years now Organized Medicine has made this possible for those in practice. This is done in several ways. Local societies and academies hold regular meetings, and either through their own members or speakers brought from University centres, or distinguished visitors from abroad and the United States, an effort is made to keep before all members of the profession the latest advances in the science of Medicine. In this work, Organized Medicine, by which is meant the Ontario and Canadian Medical Associations, has played a major part in arranging comprehensive scientific programmes in their annual meetings, and in providing speakers for any society that asks for them. To these men, drawn from the teaching faculties, the hospital staffs of non-university centres, and to distinguish visitors, the thanks of the profession at large is due and freely given. The development of this plan of postgraduate work has been going on for years

and will grow steadily in the future, so that there will be available to all practitioners a better post graduate course, costing little, and brought to their very door step.

There are however many practitioners who for one reason or another do not take advantage of the opportunity presented, to keep themselves informed of the recent advances in medicine. If these same men benefit by a form of health insurance, compulsory on the public, *is it not reasonable to demand that these practitioners be compelled to attend definite courses of instruction at stated intervals.* The British Medical Association had a scheme in preparation before war broke out whereby every five years panel doctors would have an opportunity to attend, for one month, a course of lectures, clinics, etc. During their absence from practice a locum tenens was to be provided at the expense of the Association.

It has even been suggested that a licence to practice should be good for a five-year period only, and renewal made dependent on success or failure at an examination held at the end of this five-year interval. There is much to be said for such a procedure, as all too many practitioners, even yet, read very little, if any, medical literature, go to no scientific meetings, and carry on practice at the end of 35 or 40 years with very little greater knowledge of the science of Medicine than they had on graduation. Such instruction, followed by examination, would also help to delete from our ranks the unfortunates who have fallen victim to dope and drink.

As the population of this country grows (and grow it will), we shall become more self-contained and more efficient in the matter of postgraduate studies. Within our own gates there is now in progress the "Gallie" course in surgery, and Fellowships in Medicine and Research at the Banting Institute; these are the foundation of greater efforts in this direction. This work will grow, and this University will become and be known as one of the greatest centres of Medical training, undergraduate and postgraduate.

Therefore, while it may be said of the teachers and lecturers, etc., "We do not get to know you", or, "Many cannot lecture or teach", or, "There are not adequate facilities for postgraduate instruction either in the University or outside it", take the long view; think of the way your University has developed and what has been done; look ahead; having done this, all should go forth from its halls happy and proud to call her Alma Mater.

Now may I draw your attention to another problem with which the medical profession in Canada is faced today, and for which a reasonable solution must be found: health insurance. In a matter of such importance no governments will move unless there is a known public demand for such. Education through the daily press, magazines, speeches in parliament and outside it, have created the demand. This has been strengthened since it has been made known that forty per cent of the trainees across the Dominion have been found unfit for category A. It is generally conceded that such a state of affairs must be rectified if we are to have a healthy nation. The only solution feasible seems a form of insurance whereby the main obstacle to obtaining adequate medical services, namely the economic barrier interposed between doctor and patient, can be removed.

It is the aim of the medical profession in Canada to see that any scheme of insurance that may be evolved secures for everyone in this Dominion of whatever social standing, an adequate medical service, preventive and curative; *provided that any such scheme is fair and satisfactory to all parties concerned.* With that in view every member of the profession, whether graduate or undergraduate, should study this problem, so that a well-considered, equitable plan of health insurance may be formulated and presented at the most opportune time.

Draft proposals have already been presented to the executive committee of the Canadian Medical Association, and are under consideration. A scheme was outlined by the Ontario Medical Association five years ago and is now being

studied by every medical society in the province, and revised to the end that we shall, when the occasion arises, be prepared to act as an united body to protect the interests of the public and profession alike.

Therefore I say that every undergraduate should familiarize himself or herself with the history and development of health insurance schemes in every country. Their strength and weakness, all features favourable and unfavourable, should be debated to the end that you may give an informed opinion. *It is unlikely that many graduating now or in the future will practise medicine in the way or under the conditions that obtain today.* Social revolution with all its ramifications is upon us. Let us as intelligent, educated, men and women, give it direction not only for our own protection, but for the good of all. *Always ready to live up to our best traditions in serving all, let us see to it now that we are not enslaved.* Let us make sure that the standards of education and practice are raised rather than lowered.

To that end, enroll immediately on graduation in the ranks of Organized Medicine, that, should the day of trial and testing come, we may present a united front with ranks closed. Such alliances will cost you time, effort, and money, but the reward of such investment will be a greater and more glorious profession, and a public more enlightened and better served.

The last and the most urgent subject that calls for comment is the war. Repeatedly have we been told by our responsible leaders after two and a half years of struggle, that this will be a long and bitter war. Mr. Churchill has said in effect that to win there must be consolidation, then liberation, then annihilation. Organization and consolidation of the allies' resources and manpower has just begun. Liberation has so far come only to the Ethiopians. Annihilation of the enemy is as yet a far-off goal.

That the bitter conflict may be brought to a successful issue, our armed forces need supplies, men, and more men. All the members of the armed must have medical services, preventive and curative, provided. The number of doctors needed will increase as the army increases. *Two thousand members of the profession in Canada are already enlisted in the army, navy, and air force. It is estimated that four hundred more will be needed during this year.* Hence the call to those about to graduate and to those just completing their year of hospital internship comes strong and clear. *Your duty, if physically fit and if there are no insurmountable impediments in the way, lies with the armed forces.* Courage you will need, sacrifice you will make. Those who fight and shield us with their lives must have every care and every protection that we alone as a profession can provide.

Sympathize with those in whom the "spirit is willing but the flesh weak." Pity those who without reason turn aside. *Beware of those who, when colleagues are absent on service, "break in and steal".* Go forth without fear. Farewell.

"And I said to the man that stood at the gate of the year, 'Give me a light that I may tread safely into the unknown', And he replied, 'Go forth into the darkness and put your hand into the hand of God. That will be better than a light and safer than the known way'".

HARRIS MCPHEDRAN.

Abstracts

D. P. BRYCE, Editor

Contributors { J. G. Mickler
R. J. Wilson
G. G. Caudwell

Arteriosclerosis of Coronary Arteries

Horn and Finkelstein
A.H.J. 119, 655

The study of coronary occlusion has become so important of late that an insight into the basic pathology is important. Several interesting facts arise from this study.

Firstly, although the prevalence of arteriosclerosis is perhaps slightly greater in the left anterior descending branch, yet when the arteries are broken down the incidence of occlusion is greatest in the right coronary artery.

Secondly, the role of the vascularization of the media and intima, as a result of arteriosclerosis, is emphasized in the production of acute occlusion. Intramural hemorrhage was found to lead to occlusion in 62% of the cases by

(a) inducing acute degenerative and reactive responses in the plaque overlying the hemorrhage;

(b) by obstructing the artery mechanically;

(c) by actually producing dissolution and rupture of the intimal layer which led to thrombosis.

Finally, it is impossible to postulate the pathogenesis of an occlusion from the pathology of the wall, and the only correct evaluation is as to the extent and degree of arteriosclerotic change.

D. P. B.

A New Sulphonamide (Sulphonamide E.O.S.)

By N. A. Mutch, M.A., M.D., F.R.C.P.
British Med. Journal, Oct. 11, 1941

This paper describes the properties and actions of this newly-developed drug and to summarize its content:

Sulphonamide E.O.S. is of low toxicity

and of potent chemotherapeutic activity in acute cerebrospinal meningitis in humans, and experiments upon animals lead to the probability of its effectiveness against *B. coli*, streptococcus hemolyticus, streptococcus pyogenes, pneumococcus, *B. typhosis* and *B. paratyphosis*.

Unpleasant symptoms rarely follow its use in man, and vomiting is practically unknown. Cyanosis is common but leukopenia was not observed in 130 cases treated by full dosage of the drug, for a variety of infections. Anemia has been observed only to a minor degree and drug rash is extremely rare.

Sulphonamide E.O.S. is soluble in water and thus may be given per os, per rectum or per vein. In vivo it is hydrolyzed to produce sulphonilamide and is excreted in the urine in forms far more soluble than those found with sulphapyridine or sulphathiazole, hence it is less apt to produce insoluble crystals in the renal tubules.

J. G. M.

Maternal Pulmonary Embolism by Amniotic Fluid

Steiner and Lushbaugh, Chicago
J.A.M.A., Oct. 11, 1941

The authors claim to isolate as a clinical entity several cases of maternal death during labour or shortly after, which previously have been considered as obstetric shock, post partum uterine atony with hemorrhage or perhaps as non-convulsive toxemias and acute pulmonary edemas of pregnancy. They do so by presenting eight clinical histories with a pathologic-anatomic basis and by demonstrating the production of this entity in experimental animals.

Pulmonary embolism by thrombi from

the maternal venous sinuses, or by air or placental fragments is already recognized. The authors, however, demonstrate further emboli formed from meconium, epithelial squamæ and amorphous materials of foetal origin. These findings substantiated by microscopic studies of the lungs occurred in similar clinical cases the common factor of which was difficult labour. As a further check they studied again the cases of women dying during the same period from well-known obstetrical conditions and did not demonstrate similar microscopic findings.

Although considering a rare condition, nevertheless the authors have contributed a new entity to our conception of maternal mortality.

D. P. B.

The Coronary Operation

Claude S. Beck, M.D.

The American Heart Journal, Vol. 22, No. 4

The idea of revascularizing the heart was first presented by the author in 1931, who since then has carried on over a thousand experiments on animal hearts. The results have shown that gradual occlusion of the coronary arteries was followed by development of intercoronary anastomoses. When such occlusions were being produced, the ligation of four or five small arteries of the left ventricle, distal to the origin of the main branch from the aorta, had fatal results, the ventricle going into fibrillation. When the major artery was ligated farther up and so reducing its cross-section by one-third, the heart was quite able to carry on. Although a much larger area of the myocardium was rendered ischemic in the latter case, recovery was nevertheless made, while the severe but localized ischemia, as in the former case, acted as a trigger to set off ventricular fibrillation. Consequently it was believed that anything which would improve circulation and so bring about an equal distribution of blood to all parts of the myocardium in the early stages of coronary disease, would delay or remove the threat of ventricular fibrillation. Such might be

accomplished by forming extra coronary communications by bringing in blood from outside by the use of grafts or by opening up of intercoronary channels by producing an inflammatory reaction on the surface of the heart.

In 1935, using the results of these experimental demonstrations as a basis for application to human cases, 30 patients, severely incapacitated by coronary disease and with little chance of life save that offered by the anticipated success of the operation, were chosen. The graft operation was performed, using parietal pericardium, mediastinal fat, skeletal muscle from the chest wall, lung and even omentum and spleen, brought up through the diaphragm.

The operations, with exceptions, was found to be beneficial in spite of the fact that 10 of the patients died in the immediate post-operative period. Of the remaining 20, discharged from hospital, 16 are still alive and definitely improved. The degree of benefit has been great in some cases—being capable of manual labour and quite free of symptoms; others, although not back to work, are much better. These results may be contrasted with the hopeless outlook of severe cases of coronary disease, in which recovery to a degree allowing manual labour is most unusual.

Many patients felt better as early as one week following the operation, which was completely unexpected, because cross-infection experiments had shown that such communications allowing increased blood supply to the myocardium required at least two weeks to develop. This initial improvement was believed due to the abrasion to the heart at the time of operation with the opening up of intercoronary channels. The relative value of these communications and the extra coronary anastomoses of the graft has not been determined as yet.

However, these few clinical cases give quite an inadequate appreciation of this new field and it is only by further application that a preliminary appraisal of results can be made and so determine the efficacy of the coronary operation as a therapeutical measure.

G. G. C.

The Ward Dressing

Abstract of an Editorial which appeared in *The Lancet*, Nov. 8, 1941

Infected war-wounds may be divided into two broad categories: (1) wounds infected outside the hospital ward which are for the most part infections with the anaerobic sporing bacilli, clostridium telani and the gas-gangrene group; (2) wounds infected in the hospital ward when the principal pathogens are the pyogenic bacteria staphylococcus and streptococcus and the coliform bacilli, including proteus and pyocyanus. These pyogenic organisms are found in a very small proportion of open wounds examined on admission to hospital—approximately five to six per cent in the present war and fifteen to twenty per cent in the last. After a week's stay in hospital, the proportion of infected wounds may exceed fifty per cent and may increase with further residence to seventy to ninety per cent. The streptococcus is the most dangerous of the pyogens by virtue of its great invasiveness, but the coagulase-positive staphylococcus which in the past year has been the most frequent pathogen, may also produce much chronic invalidism if it gain access to bone. It is true, and perhaps unfortunate, that infection produces little evidence of local or systemic upset in many wounds, particularly if they are shallow and draining well, and does not greatly retard healing, so that a complacent surgeon may regard such infection as innocuous. But once in a while an unfortunate "accident" occurs in that a new patient develops cellulitis or septicaemia, signs of osteomyelitis, in a case of compound fracture; nurses go off duty with septic fingers and sore throats, and the surgeon realizes too late that infection is rampant in the ward.

Certain teams of surgeons and bacteriologists devoted their time and energy in the past winter to researches designed to solve the problem of preventing these tragic consequences of wound-infection. Their findings have been published in a memorandum entitled *The Prevention of "Hospital Infection" of Wounds*, which

presents the problem of hospital infection of wounds and practical instructions for its prevention. If it be granted (and the circumstantial evidence is very strong) that pyogenic infection of open wounds takes place mostly in the hospital ward, the possible sources must be defined and controlled and the channels of spread blocked. The principal sources are:

1. Wounds already infected.
2. Noses and throats of patients and ward personnel.
3. The dust of the ward which has become infected from the first two sources.

The channels of spread are:

1. Direct transference by hands, instruments and dressings which become bacterial carriers from contact with already infected wounds or respiratory tracts.

2. Droplet infection from the nose or mouth of the attendant or patient (5 to 10% of adults are throat carriers of *Strep. pyogenes*; 30 to 40% are nasal carriers of *Staph. aureus*).

3. Air currents carrying infected dust or droplet-nuclei. With the increase of infected wounds, dust and air become more heavily infected, sore throats appear and a vicious circle sets in.

It may well be, although the point has not been adequately proven, that direct transference by infected hands or instruments is more important than droplet or aerial infection. The latter two channels can be, for the most part, blocked by efficient masking of all ward personnel (if pains are taken to introduce measures of this type) and by the maintenance of silence among the patients during the period of dressings. Further, and of no little importance, a still atmosphere must be attained by completing bed-making, sweeping and dusting at least an hour before dressings are begun. Windows and doors should be kept closed to minimize air-currents; movement of bed-clothes must be done gently and slowly, and floors may be treated with an oil preparation to adsorb the dust.

The memo describes in detail a technique for the control of contact infection in ward-wound dressing. This may

entail a radical revision of procedures at present carried out in many places. The key-points are:

1. That the hands of the dresser are to be regarded as at all times infected.

2. The wound and the surrounding skin are to be likened to a plate of culture medium—that is, a surface very susceptible to infection and therefore to be uncovered for the shortest possible time and as infrequently as possible.

GONE ARE THE DAYS WHEN WOUNDS ARE GAILY LAID BARE FOR THE ROUND OF INSPECTION BY THE BIG "WHITE CHIEF" AND HIS RETINUE OF MASKLESS FOLLOWERS.

Dressings are carried out by a dressing team, each member of which is well-versed in his or her own duties and each wound must be inspected and re-dressed before the next is dealt with. The team consists of the dresser (who is preferably the house-surgeon or someone equally competent), the trolley-attendant, a trained nurse who is responsible for the care and sterility of instruments, dressings, bowls and so on, and a "dirty assistant" to lend a helping hand in removing bandages or holding a limb and to handle the lids of receptacles for discarded dressings after the outer dressing has been removed by hand. The inner dressing is changed by the dresser with long instrument-forceps in hands that are washed and dried but not "scrubbed up." Every step is designed to prevent the transference of infective material from one wound to another by hands, instruments, or dressings. Details are included regarding the sterilization of instruments and receptacles. Practical instruction is given regarding the irrigation of wounds, chemical disinfectants, masks, sterilization, and cleansing of baths. Attention is directed to the danger of cutting and changing plasters in the ward, for plasters usually are heavily contaminated with bacteria both inside and out.

It is recommended that nurses be given a *practical* course in bacteriology which will emphasize to them the importance of infection far better than will a series of abstruse lectures. For their intelligent co-operation is absolutely es-

sential for successful prevention of cross-infection in wards.

If the recommendations in this pamphlet seem to some surgeons rather elaborate with an air of the laboratory about them, let them not on that account dismiss them as impracticable or unnecessary. Infection of open wounds is a menace to be constantly guarded against and it is utterly illogical to use a less strict technique in dressing these susceptible wounds in the ward than is used for less vulnerable tissues in the operating theatre. The practicability and effectiveness of this new technique has been shown by McKissock, Wright, and Miles, who reduced the incidence of hospital infections in air-raid casualties from 31.3 to 2.2% in improvised E.M.S. wards. Local sulphonamide prophylaxis and other new methods may help to minimize hospital infection of wounds, but the soundness of the principles enounced in this memo regarding the dressing of open wounds cannot be over-emphasized. It should be read and digested by every surgeon, house-surgeon, medical student, hospital bacteriologist and surgical ward sister.

R. J. W.

Haemolytic Streptococci in Normal People and Carriers

Ronald Hare

The Lancet, January 18, 1941, p. 85

As a result of extensive bacteriological investigations, it was concluded that the haemolytic streptococci which cause infection of wounds in both civil and military life are of exogeneous origin and are transferred to the wound some time after its infliction. The group of haemolytic streptococci which is pathogenic for human beings—group A—is probably not represented among the micro-organisms usually associated with the majority of objects which cause wounds or with substances which may find their way into wounds at the time of infliction. However, group A strains in the nasopharynx of attendants or about their clothing and hands or in other haemolytic streptococcal infections in patients in the near

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¹Borsook, H., "Vitamins", Viking Press, 1940.

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neighborhood frequently have access to the wound while it is exposed for inspection or for dressing.

From an analysis of the data accumulated during the present investigation it was found that the presence of haemolytic streptococci—group A—on the hands or skin of the general population (which in this instance was represented by two series of students studying pre-clinical subjects) was extremely uncommon. The only person with these organisms on his hands in the present investigation of 248 persons had almost certainly picked them up by contact with an active infection. It has been suggested that infrequent bathing might bring about an increase in the number of streptococci on the skin and thus be in part responsible for the high incidence of streptococcal infection of wounds. In the investigation of the bacterial flora from an area of skin left unbathed for two weeks, streptococci of other groups were acquired but no group A strains were found. Swabs from the clothing of 121 persons failed to reveal the presence of group A haemolytic streptococci.

From studies of known nasopharyngeal carriers, it was found that there was a distinct tendency for the hair, the skin of the face and hands and the pocket in which the handkerchief was kept, to be contaminated. Experiments showed that group A haemolytic streptococci kept at room temperature might survive on clothing for a period of 30 to 40 days.

Examination of road dust and soil taken from roadways and paths along which pedestrians and vehicles constantly passed and which were accessible to human contamination failed to reveal the presence of haemolytic streptococci group A.

Examinations were conducted in the rooms inhabited by carriers during the acute phase of their illness and during the succeeding period while they were under investigation. Haemolytic streptococci were found on bedclothes, carpets, etc., about the room. These findings are in accord with those of other workers who showed that dust of wards in which infected burns, puerperal infections and scarlet fever are being nursed

may be heavily contaminated with haemolytic streptococci—group A.

R. J. W.

Cardiac Neuroses

By John Oille, Toronto

Sixty per cent of patients who consult a cardiac specialist are suffering from a cardiac neurosis. Some are ready to describe at length their fears and symptoms, others let it slip, while others hide it very well and are only suspected by the relief they express following an examination. The usual source of the neurosis is an ill-advised remark by their doctor which acts upon a sensitive suggestible pessimistic type of individual. It must be remembered that actual heart disease may exist in these people. There are several types of pain.

A. Sub-mammary, i.e. between left nipple and costal margin. When confined here it is almost never cardiac in origin. May be due to cholecystitis or a root neuritis.

B. Little shoots or stabs of pain, probably never cardiac and usually with no demonstrable cause.

C. Root neuritis from spondylitis, which pains vary in location, character, and duration too greatly to be cardiac in origin.

D. Cardiospasm and heart-burn simulate cardiac pain closely.

E. The "globus", lasts a long time and follows worry and fatigue.

F. Epigastric pain should not be considered cardiac unless it radiates.

G. "Soldiers' " heart or "effort synchome", frequently have pains about the heart which are probably not cardiac.

Sympathy, interest, encouragement, and explanation of the symptoms are important in the treatment.

Blood pressure is a common source of anxiety and these neuroses are often created by ill-advised advice; this is particularly true of "hyper reactors", all of which do not advance to persistent hypertension.

Palpitation frequently worries people and they should be reassured. This, too,

is usually normal. A diseased heart must of course be ruled out first.

Many a patient has been worried by being told he has a leaking valve or a flabby heart, etc., when frequently the murmur is unimportant. Extra systoles are a common cause of worry and often sighing or the occasional deep breath is mistaken for shortness of breath or puffing.

Incorrect diagnosis of aneurism of the aorta misinterpreted dizziness, weakness,

and numerous other apparent trifles have incapacitated many needlessly.

In dealing with these cases it is important to be a good listener and very tactful so as not to lose their confidence. Reassurance and encouragement must be emphatic and dogmatic after explanation of their symptoms.

Patients with real heart disease nearly always have exaggerated anxieties, and reassurance here is just as necessary.

D. P. B.

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